





EVALUATION OF VARIABLES AFFECTING LOWER EXTREMITY  
ALIGNMENT IN THE AETIOLOGY OF PATELLOFEMORAL PAIN  
SYNDROME

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*The courage to change what should be changed,  
The serenity to accept what cannot be changed,  
And the wisdom to distinguish the one from the other  
(R. Niebuhr)*



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# **CHAPTER 1**

## **GENERAL INTRODUCTION**



## **GENERAL INTRODUCTION**

The patellofemoral joint (PF-joint) is, in relation to its size, one of the most described joints of the human body. The reason for this is attributed to the controversies that exist concerning the aetiological mechanisms which are thought to be responsible for dysfunctions that may occur at this joint. The patellofemoral dysfunction syndrome (PFDS) is one of the most common knee disorders seen in physically active individuals.(10,18,22,28,37,66,77,88) The literature describes an incidence of one in four in the general population, and even higher among athletes. (3,57,122) The high incidence of patellofemoral pain (PFP) indicates that this pathology can be identified as the most important cause of knee problems and pain in a wide range of physically active individuals. As a result of an increased participation of the public in sports during the recent decades, a proportional increase in the amount of patients suffering from PFP can be noticed in players practising ball games, cyclists and in runners.(19, 77, 101, 109,112,114)

### **1. Definition of patellofemoral pain (PFP)**

In the literature there seems to be no clear consensus regarding the terminology for pain in the anterior aspect of the knee. Patients experience a variety of symptoms comprising pain which is difficult to define at various locations and levels, resulting in different degrees of physical impairment.

Until the end of the 1960s patellofemoral pain was attributed to chondromalacia patellae because till then the general opinion ruled that anterior knee pain was caused by softened patellar articular cartilage. The term chondromalacia patella (CMP) was used in the literature for the diagnostic description of all unclear and diffuse forms of anterior knee pain. However, in 1976, Insall propounded to use the term CMP only in those cases in which there is objective evidence of a lesion of the patellar cartilage.(52) In the mid 1970s, Insall's opinion was followed by other authors who found a poor correlation between the clinical findings of anterior knee pain and the presence of patellar articular changes in arthroscopic studies.(23,65,93) Leslie and Bentley reported that only 51% of patients with a clinical diagnosis of chondromalacia patellae had changes on the patellar surface when they were examined by arthroscopy.(65) Royle et al. analyzed 500 arthroscopies, performed in a 2-year period, with special reference made to the patellofemoral joint. They concluded that patients with patellofemoral pain do not always have patellar articular changes, and patellar pathology

is often asymptomatic.(93) In agreement, Dye could not provoke any pain during arthroscopic palpation of his extensive lesion of the cartilage without intra-articular anesthesia.(23)

According to the International Patellofemoral Study Group (IPSG), the term chondromalacia patellae should not be used to describe a clinical condition but is merely a descriptive term for morphologic softening of the patellar articular cartilage and is not synonymous with patellofemoral pain.(53) Since there is evidence that patellofemoral pain in essence is caused by a dysfunction of the patellofemoral joint with overuse of the osseous and/or surrounding soft tissues, with or without the presence of patellar articular changes, today it is the general opinion to use the term patellofemoral dysfunction syndrome (PFDS). (9,29,110) Pain represents the symptom that the majority of patients experiences. Patients report retropatellar pain during and after activities such as running, squatting, kneeling, going up and down stairs and hills, cycling, jumping, pain during and/or after prolonged sitting with the knee in flexion (“movie sign”) and rising from a seated position. The pain is typically dull and diffuse around the patellofemoral joint. Besides pain, patients often report instability of the knee. Patients may complain of giving way of the knee as a result of reflex inhibition of the quadriceps muscle secondary to pain, effusion or deconditioning. Giving way due to patellofemoral pain typically occurs during ascending or descending stairs or an incline. PFP symptoms may arise after a direct trauma to the knee but usually have an insidious onset caused by overuse, which could be a new activity or an increase in duration, frequency or intensity of the patient’s (sports)activities. (16,62,122) PFP-patients often also report symptoms other than pain or instability such as sensations of stiffness and swelling, locking and crepitus at the knee.(8,36,107) Therefore, it seems appropriate to use the word ‘syndrome’ defining a group of symptoms and signs that occur in a combination and characterizes a particular abnormality.(103)

## **2. Aetiology of PFP**

Prevention of the onset of patellofemoral pain is a major goal for many sports medicine practitioners. Before an approach in planning and carrying out the prevention and treatment of patellofemoral pain can be set up, understanding of the aetiology associated with the patellofemoral dysfunction syndrome is essential. In the 1990s, Dye introduced the tissue homeostasis theory concerning the aetiology of patellofemoral pain.(24) He states that the function of the patellofemoral joint (and any other joint) can be characterized by a load/frequency distribution (the envelope of function) that defines a range of painless loading that is compatible with homeostasis of the joint tissues. According to Dye, the “envelope of

function” describes a range of loading/energy absorption that is compatible with tissue homeostasis of an entire joint system, that is, with the mechanisms of healing and maintenance of normal tissues. If an excessive loading is placed across the joint, loss of osseous and soft tissue homeostasis can occur resulting in pain. Risk factors which cause an internal load shifting within the patellofemoral joint may lower the threshold (i.e. decrease of the envelope of function) for the loss of tissue homeostasis leading to the perception of patellofemoral pain.

In view of the high frequency of patellofemoral problems, as well during leisure time activities as in professional sports, it is clear that analyses of these risk factors for patellofemoral pain is urgently required. This refers to information on why a particular individual may be at risk in a given situation and another individual, exposed to more or less the same exercise load, does not. In order to answer the question why a patient develops loss of homeostasis of patellofemoral joint tissue, risk factors for the development of patellofemoral pain syndrome need to be identified.

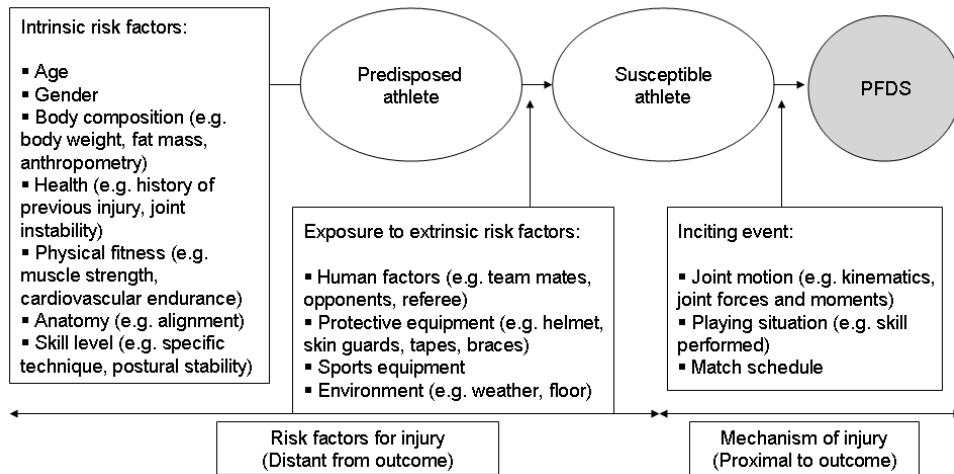
### *2.1. Multifactorial model of PFP aetiology*

In the literature general consensus exists that the patellofemoral dysfunction syndrome is associated with multiple causative factors and can be considered as a multifactorial problem with a possible interaction of multiple risk factors at a given time.(16,41,76) Although the exact aetiology of the patellofemoral dysfunction syndrome remains unclear, as stated above, in the majority of the cases the aetiology is probably multifactorial, resulting from a combination of risk factors, which can be divided into intrinsic and extrinsic risk factors.(28,77,78,105,113)

Extrinsic risk factors are related to factors outside the human body, such as the type of sports activity, exercise load and intensity, amount of physical activity, equipment, weather conditions, environmental conditions.

Intrinsic risk factors relate to individual physical and psychological characteristics such as age, gender, conditioning, muscle strength, muscle tightness, muscle imbalances, joint stability, biomechanics, etc.

Meeuwisse presented a model which describes how multiple factors can interact to produce an injury (figure 1).(76) This model shows that numerous intrinsic risk factors may predispose an individual to the development of patellofemoral pain.



**Figure 1.** Dynamic, multifactorial model of sports injury aetiology (Adapted from Meeuwisse.)(76)

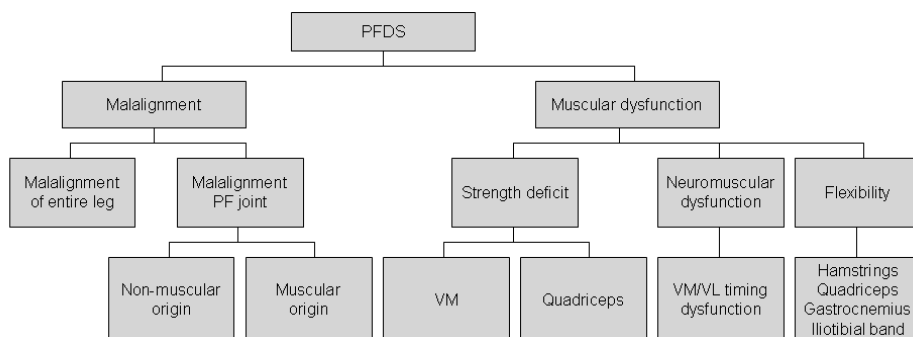
Although the onset of the patellofemoral dysfunction syndrome can be the result of an acute trauma, patellofemoral pain is usually caused by a dysfunction of the patellofemoral joint resulting in an overuse of the joint.(69) This overuse of the patellofemoral joint can be the result of a variety of reasons but it is an accepted fact that it arises from a discrepancy between the load on (extrinsic risk factors) and the load tolerance of the patellofemoral joint, which is determined by the various intrinsic factors.(78,122)

## 2.2. Intrinsic Risk Factors

Regarding the intrinsic risk factors of PFP, in the literature an abnormal tracking of the patella in the femoral groove during flexion and extension of the knee joint is considered to be the most important aetiological mechanism for the development of PFP.(25,33,40, 51,73,74,75,124) Imperative for a normal tracking of the patella in the trochlear groove of the femur is a good functioning of the static (non-muscular) and dynamic (muscular) stabilisers of the patellofemoral joint. Malfunctioning of these static or dynamic stabilisers results in maltracking of the patella in the femoral groove, which significantly decreases the load tolerance of the patellofemoral joint and thereby increases the risk of patellofemoral overuse.

Numerous intrinsic factors have been identified as potential risk factors for the development of patellofemoral pain.(12,86,122) These intrinsic risk factors can be divided into alignment problems within the patellofemoral joint and outside the patellofemoral joint.(125) Considering the origin of possible malalignment, a distinction can be made between muscular structures (dynamic stabilizers) and non-muscular structures (static stabilizers) (figure 2).





**Figure 2.** Clinical classification of PFDS (Adapted from Witvrouw et al.) (125)

### 2.2.1. Static stabilisers PF-joint

Bony abnormalities such as an osteochondral dysplasia of the femoral trochlea, with a shallow or even a convex trochlear groove and hypoplasia of the medial patellar facet have been identified as possible causes of patellofemoral malalignment relating to patellofemoral pain and instability. Also retinaculum dysfunctions, such as tightness of the lateral retinaculum leading to hypomobility of the patella puts excessive stress on the lateral patellofemoral joint as a result of lateral patellar tracking and tilting. (32,43,47,90,99,119,125) Dysfunction of the retinaculum can also result in a hypermobile patella. Research has revealed that an increased medio-lateral patellar mobility is a risk factor for patellofemoral pain.(120)

### 2.2.2 Dynamic stabilisers PF-joint

Dysfunction of the muscular structures implies muscle strength, neuromuscular condition and muscle flexibility (figure 2).(119,125) Decreased knee extensor strength is common in patients with PFP. Dysfunction of the m. Quadriceps not only comprises a weakening of the whole quadriceps muscle but various patterns of quadriceps muscular dysfunction have been reported such as quadriceps hypotrophy, selective vastus medialis (VM) hypotrophy and a neuromuscular timing dysfunction between the vastus medialis and vastus lateralis (VL).(103,117,125)

Retrospective studies have reported a significant loss of quadriceps strength in patients with PFP. This strength deficit was specifically observed during eccentric contractions of the quadriceps.(4,108,118) In a prospective study, Witvrouw et al. also identified a decreased explosive strength of the quadriceps as a risk factor for the development of PFP.(122) Muscle-strength deficits of 15% or more than the controlateral asymptomatic limb are considered to be abnormal.(125)

Selective hypotrophy of the vastus medialis (VM) has also been shown to be a common clinical finding in PFP-patients. Several studies have reported that the EMG VM/VL ratio in PFP-patients is less than in healthy subjects, with a decrease in VM activity.(9,14,80,87,100) The lower activity of the vastus medialis related to the activity of the vastus lateralis (VL) could lead to an imbalance between the VM and VL which is considered to be closely linked to patellar maltracking and the development of patellofemoral pain.(71)

Besides a strength deficit of the entire quadriceps muscle and a selective VM hypotrophy, a disturbed neuromotor control of the patellar agonists has been found to be associated with the development of PFP.(117,124) Witvrouw et al found significant differences in reflex response times between the vastus medialis and the vastus lateralis in patients suffering from PFP.(124) It was hypothesised that in some patients with PFP the VL contracts earlier than the VM, contributing to a laterally directed force on the patella.(124)

In addition to strength and neuromuscular dysfunctions of the quadriceps muscle, loss of flexibility of the quadriceps and other muscles closely related to the knee have been found to have a profound effect on patellofemoral joint biomechanics.(27) Witvrouw et al. identified a tight quadriceps muscle as a risk factor for the development of PFP.(122) Tightness of the lateral muscles such as the tensor fascia lata and iliotibial band is also associated with PFP.(21) A tight iliotibial band causes a lateral tracking and tilting of the patella and often a weakness of the medial retinaculum as it pulls posteriorly on the lateral retinaculum when the knee flexes.(32,35) Tightness of the hamstrings has been stated to necessitate greater force being generated by the quadriceps and consequently leads to an increase in patellofemoral joint reaction force. (27)

### 2.2.3 Malalignment outside the PF-joint

During the past decades, many research has been done and interventions have been proposed which have been focussing on the patellofemoral joint itself, with the intention to influence patellar motion (e.g. strengthening of the vastus medialis, stretching, patellar taping and bracing, soft tissue mobilisation, patellar mobilisation). However, it has been recognised that the mechanics of the patellofemoral joint are also influenced by segmental interactions of the lower extremity.(22) Consequently, in the more recent years there has been a growing interest in the repercussions of dysfunctions and altered kinematics in the distal and proximal links of the patellofemoral joint within the kinetic chain, namely at the foot, ankle and hip joint, on the biomechanics of the patellofemoral joint. Abnormal motions of the tibia and femur in the

transverse and frontal planes are believed to have an effect on patellofemoral joint mechanics and patellofemoral pain.(88)

A biomechanical factor of the PF-joint which is thought to be highly subjected to impaired biomechanics and postures of the leg is the Quadriceps angle (Q-angle). The Q-angle is a clinical measure of the alignment of the quadriceps femoris muscle relative to the underlying skeletal structures of the pelvis, femur and tibia and is used to reflect the quadriceps femoris muscle's force on the patella in the frontal plane.(97) The angle is formed by the imaginary line from the anterior superior iliac spine to the centre of the patella and from the centre of the patella to the middle of the anterior tibial tuberosity.(67,97) A normal Q-angle is reported to be 12° in males (ranging from 10° to 14°) and 15° in females (ranging from 14.5° to 17°). (97,119).

Aberrant postures and dysfunctions at the leg such as anteversion of the hip, external tibial torsion, genu valgum, tightness of the tensor fascia lata and of the iliotibial band, gluteus medius weakness and pronated feet have been indicated to cause an increase of the Q-angle.(96)

An increased Q-angle may predispose the patella to excessive lateral tracking and stress.(97) A Q-angle which exceeds 15-20° is thought to contribute to knee extensor dysfunction leading to patellofemoral problems.(68) This hypothesis is supported by a retrospective study of Messier et al, who found that the Q-angle was a strong discriminator between runners suffering from patellofemoral pain and non-injured runners.(77) However, others have questioned the correlation between an increased Q-angle and the development of PFP.(5,13,28) Nonetheless, it has been stated that the Q-angle should be regarded as one bit of information, which might correlate with other clinical findings in order to understand the malalignment problem as fully as possible.(32)

Based on previous studies in the literature it can be assumed that, besides risk factors for PFP which directly influence the patellofemoral joint, dysfunctions and alterations in the alignment of the proximal and distal segments of the lower extremity with respect to the PF-joint, namely at the hip, ankle and foot, may also influence the alignment of the PF-joint. Indeed, according to the kinetic chain theory, biomechanical changes in a specific joint may result from dysfunctions and altered kinematics in the joints laying proximal or distal to this joint. Dysfunctions and alterations in the alignment of the foot and hip may consequently result in malalignment of the PF-joint and possibly lead to PFP.

The evaluation of variables, affecting the alignment of the segments laying proximal and distal to the PF-joint in the kinetic chain, therefore is important for the investigation of

possible sources beyond the patellofemoral joint, which may lead to the development of patellofemoral problems.

Looking at the segments distal to the patellofemoral joint, intrinsic imbalances of the ankle and foot, with a focus on an abnormal foot pronation and subsequent lower-extremity rotation, have been propounded to be predisposing factors for the development of PFP.(26,46,59,70,109) This hypothesis has been supported by Lun et al. who identified that forefoot varus was a potential risk factor for PFP in a group of recreational runners.(70) Eng & Pierrynowski revealed that the use of orthotics, correcting excessive pronation of the foot, in conjunction with an exercise programme was more effective than an exercise programme alone in patients with PFP.(26) Corresponding with this, Klingman et al. noted a significant change in patellar positioning (medial glide) after the use of semi-rigid medial rearfoot-posted orthotics.(59) However, Powers et al. did not find significant differences in the magnitude and timing of peak foot pronation between individuals with and without patellofemoral pain.(86) Also, Hetsroni et al. did not find a consistent association between static or dynamic parameters of foot pronation and the risk of PFP.(48) Duffey et al. demonstrated that runners suffering from PFP had 25% less pronation during the first 10% of the support phase of the foot during running.(22) Consequently, in the literature there seems to be no clear consensus concerning the role of abnormal static or dynamic foot pronation as a risk factor for the development of patellofemoral dysfunction.

Considering the segments proximal to the patellofemoral joint, the kinetic chain theory suggests that proximal core hip strength is needed for control of distal segments.(82) The hypothesis that the force of the muscles surrounding the hip might play an important role in controlling the movement of the knee in the frontal and transversal plane has been addressed by Ireland et al.(54) In their study, these authors found that individuals with patellofemoral pain had 26% less hip abduction strength and 36% less hip external rotation strength than similar age-matched controls and they theorized that hip muscle weakness may result in uncontrolled femoral adduction and internal rotation leading to an increase in the dynamic Q-angle, contributing to patellofemoral joint stress. Similar findings are reported by Robinson et al.(92) However, although both authors concluded that hip muscle weakness might predispose the patella to lateral tracking, neither investigation examined hip or knee kinematics. Consequently, it is unknown whether the subjects actually demonstrated excessive hip adduction, hip internal rotation and knee valgus during a dynamic activity. Mascal et al. investigated the interrelationship between hip strength and hip kinematics in one single

subject with PFP and showed improved hip strength and kinematics (less hip internal rotation and hip adduction) following a 14-week strengthening intervention of the hip, pelvis and trunk musculature.(72) Although this result provides preliminary evidence to support the theory regarding hip muscle weakness and altered lower extremity kinematics, the authors did not examine knee kinematics, so it remains unclear what effect hip muscle weakness has on knee valgus.

Therefore, it can be concluded from the literature that hip muscle weakness seems to be associated with impaired biomechanics and postures of the leg that may contribute to the development of PFP, however additional studies that simultaneously examine hip and knee strength and kinematics are advised to better understand this interrelationship.

### **3. Imaging of patellofemoral disorders**

Imaging of patellofemoral disorders is considered in the literature as a diagnostic step in the diagnosis of PFP, which, together with the history and physical examination can confirm the clinical impression of PFP in the evaluation of the patient presenting this pathology. (128) According to the literature, imaging of patellofemoral pathology is usually performed by taking radiographs, computed tomography (CT) or magnetic resonance imaging (MRI). (25, 62, 129) Standard radiographic views are suggested to include standing anteroposterior view, a lateral view with the knee in 30° of flexion, and an axial view with the knee in 30° of flexion. (62, 128) Weight bearing anteroposterior radiographic views allow to evaluate varus, valgus, and degenerative changes in the tibiofemoral joint. The lateral view allows evaluating the patellar height: patella alta or a patella baja. Patella alta is considered to be a predisposing factor for PFP because it causes higher patellofemoral stress as a result of a reduction in contact area between the patella and femur. (130) Axial radiographic views provide the most information about the PF-joint and are indicated to be useful for detecting patellar and trochlear dysplasia. (62) The sulcus angle measures the depth of the trochlea and has proven to be a reliable indicator of trochlear dysplasia. (131) A sulcus angle greater than 142° indicates global dystrophy of the trochlea, which has been indicated to be frequently associated with patellofemoral problems.(32) Axial radiographs are also used to evaluate patellar position and tilt and are useful to identify changes typically seen with the lateral patellar compression syndrome, a condition that has been associated with PFP. This condition occurs when a very tight lateral retinaculum causes excessive stress on the lateral PF-joint. A tilt angle of the patella, which is formed by the angle between the line along the articular

surface of the lateral patellar facet and the line that runs across the apices of the femoral condyles, of less than 8° is considered to be abnormal. (129)

Conventional axial radiographs cannot image the PF-joint clearly at flexion angles less than 30°. However, there are subtle cases of patellofemoral malalignment, which manifest themselves at the first degrees of knee flexion, in which the diagnosis is impossible by conventional radiology. CT allows to evaluate patellar tracking from 0° to 30° of knee flexion. (128) A CT scanning measurement, which is considered to be essential in the imaging of patellofemoral disorders is the TTTG (Tibial Tuberosity-Trochlear Groove) measurement. The relationship of the position of the tibial tuberosity to the trochlear groove will determine the lateralisation force acting on the patella through quadriceps contraction. This relationship can be evaluated by the TTTG measurement. This measurement evaluates the distance (in mm) between two perpendiculars to the bicondylar axis. One perpendicular passes through the centre of the tibial tuberosity and the other through the centre of the trochlear groove. (133) The measurements are taken by superposing two CT scan cuts, one cut at the level of the proximal third of the trochlear groove and the other at the superior part of the tibial tuberosity. A TTTG distance of less than 20 mm is considered to be normal. (134) This measurement has been indicated to be abnormal in 56-93% of cases with patellar instability and nearly all cases with trochlear dysplasia. (132)

The remark has to be made, however, that the above described imaging procedures give a static view of the PF-joint, while patellar tracking is a dynamic process. Kinematic and dynamic axial MRI is currently believed to be the best tool available for analyzing patellar tracking, as it takes into account muscle contraction, movement and loading. (62) Kinematic MRI has demonstrated significantly more patellar tilt and lateralisation during quadriceps contraction at 0°, 10° and 30° of knee flexion in subjects with PFP compared with asymptomatic controls. (135)

#### **4. Conservative treatment of PFP**

Nowadays, it is generally accepted that patients with PFP should initially be treated by non-operative means. Only in patients with severe functional complaints, who do not respond to careful long-term conservative management, surgery might be considered.(36,39) Nonoperative treatment of PFP has been described to be succesfull in 75 to 84% of the cases.(21) Conservative treatment of PFP can include: rest, use of nonsteroidal anti-inflammatory medications (NSAIDs), physical therapy, shoe orthoses and knee braces.

Rest from irritating activities, particularly running and jumping, has been shown to reduce patellofemoral pain. Milgrom et al. stated that thirty percent of patellofemoral pain caused by overuse resolves after 4 weeks of decreased activity.(79)

NSAIDs are commonly used along with physical therapy in the treatment of PFP, however, today there is still insufficient evidence for their efficacy. (7)

By balanced strengthening, muscle stretching and proprioceptive training, physical therapy exercise programs aim at balancing the forces acting on the patella, with the goal of improving patellar tracking and thereby decreasing patellofemoral contact stress.

Many physical therapy protocols emphasize on strengthening the vastus medialis for its presumed medial stabilising effect on the patella. The VM is believed to prevent lateral patellar subluxation by pulling the patella medially during knee extension and therefore several authors have propounded that the function of the VM is imperative for proper patellofemoral tracking. (87, 119, 124) Selective hypotrophy of the vastus medialis is a common clinical finding in PFP patients. Consequently, numerous exercises have been proposed in the literature as exercises to selectively strengthen the VM. However, no studies have been able to confirm that the VM can be selectively strengthened by exercises.(14,121,136,137) Some investigators have proposed selective strengthening of the VM by incorporating hip internal rotation and adduction during quadriceps strengthening exercises.(21,49,63) Laprade et al., however, did not find a greater recruitment of the VM compared with VL during hip adduction or an combination of hip adduction and knee extension. (136) Mirzabeigi et al. investigated nine exercises, including isometric knee extension with the hip at neutral, 30° external, and 30° internal rotation, isokinetic knee extension through full range, and in the terminal 30°, sidelying ipsilateral and contralateral full knee extension, and stand and jump from full squat, in an attempt to discover a particular exercise useful for selectively strengthening the VM. (137) The results of their study however showed that for none of the exercises the activity of the VM was significantly greater than the VL activity. Cerny also examined several open- and closed-chain quadriceps strengthening exercises but also concluded that neither exercises were able to selectively activate VM activity. (14) Presently, the only known possible way to selectively strengthen the VM is by use of electrical stimulation. (138) Werner et al. demonstrated that a 10-week treatment of electrical stimulation of the VM, combined with stretching of lateral thigh muscles, was beneficial in two-thirds of the treated PFP patients, regarding pain and return to athletic activities and this improvement remained at 3,5 years follow up.(138)

Until approximately twenty years ago open kinetic chain (OKC) leg extension exercises have been the traditional means of strengthening the quadriceps. (18,61) Several authors, however, have reported that OKC exercises may exacerbate symptoms in patients with PFP.(20,73) In the more recent years, closed kinetic chain (CKC) exercises have been commonly recommended, because CKC exercises are presumed to be safer than OKC exercises since the former place minimal stress on the PF-joint in the functional range of motion.(95,102,126) Another reason why these exercises have received increased attention is that they simulate and replicate many functional movements. Since studies have demonstrated that the major changes as a result of strength training are task specific, several authors have proposed that it may be better to incorporate the rehabilitation into task-related practice.(84,94) Tang et al showed that in CKC exercises, there is more selective VM activation than in open chain exercises.(106) Stiene et al found CKC exercises to be more effective than OKC exercises in restoring perceived function in patients with PFP.(103) However, other researchers have stated that concerning the use of CKC exercises versus OKC exercises, any one program of quadriceps strengthening is not more effective than the other.(7) Witvrouw et al showed that, both, open and closed kinetic chain exercise programs lead to an improved subjective and clinical outcome in PFP-patients.(123) Despite the fact that in their study a CKC exercise regime showed to be slightly more effective in reducing pain and improving functionality compared to the OKC exercise regime, the authors recommend the use of both closed and open kinetic chain exercises in the treatment protocol for patients with PFP.

Weakness of the hip muscles, and more specifically of the hip abductors, external rotators and hip flexors, has been documented in individuals with PFP compared with pain-free controls. (54,111) Hence, training of the gluteus medius and iliopsoas muscle has been propounded to be necessary to improve pelvic stability and to decrease hip internal rotation and the consequent valgus vector force that occurs at the knee.(72,111) In a case report, Mascal et al described the treatment of two PFP-patients, demonstrating excessive hip adduction, hip internal rotation and knee valgus during gait and a step-down manoeuvre, focussing on recruitment and endurance training of the hip, pelvis and trunk musculature.(72) The authors showed that both patients experienced a significant reduction in patellofemoral pain, improvement in gluteus medius and gluteus maximus force production and hip kinematics and were able to return to their original levels of function. Tyler et al demonstrated that improvements in hip flexor strength combined with increased iliotibial band and iliopsoas flexibility were associated with excellent results in patients with PFP. (111)



Exercises to improve proprioception are also commonly included in the conservative treatment protocol of PFP since an abnormal proprioception has been demonstrated in PFP-patients.(2,56) In addition, it has been suggested that proprioceptive input seems to contribute to the neuromuscular control of patellar tracking.(115)

As a decreased flexibility of the m. quadriceps, m. hamstrings, iliotibial band, and m. gastrocnemius are considered to be potential risk factors for PFP, in addition to exercises aiming at improving muscle strength, neuromuscular control and proprioception, stretching of the m. quadriceps, mm. hamstrings, m. gastrocnemius and iliotibial band is considered to be an important aspect of the conservative treatment of PFP. (21,104)

Even though there is controversy between studies concerning an association between excessive subtalar pronation and the incidence of PFP, orthotic shoe inserts have been shown to decrease pain in patients who have excessive subtalar pronation.(26) Orthoses have been reported to reduce maximum pronation velocity, time to maximal pronation, and total rearfoot motion during walking and running.(89) They also appear to limit the internal rotation of the tibia and the Q-angle at the patellofemoral joint, which, theoretically, reduces the laterally directed resultant forces of the soft tissues and the contact pressure of the patella on the femoral condyles.(44) In this way orthoses have been supposed to contribute to the alignment of the patellofemoral joint.(60)

Taking into consideration the arsenal of treatment options which are available for the conservative management of PFP, the treatment protocol of PFP-patients should be based on findings from the patient's history and clinical and functional assessment.(125) Different patients, diagnosed with PFP, may present with different symptoms and signs, which makes a flexible approach necessary. Therefore it is stressed in the literature that a thorough evaluation and assessment will reveal each patient's set of clinical signs and the treatment of PFP-patients should be individualized. (36,125)

In addition to interventions which have been proposed with the intention to influence patellar and lower limb kinematics, numerous reports exist on therapeutic interventions associated with the use of braces as a conservative treatment for PFP.(11,30,42,81,98,110) The mechanical function of those braces is believed to improve patellar tracking and maintain the patellofemoral alignment by stretching tight lateral structures (lateral retinaculum). In addition, knee braces have been advocated because in a number of patients their application

causes a reduction in pain, which makes a more rapid progression of an exercise program possible.(73) It has been suggested however that besides this mechanical function braces may be effective in the treatment of patellofemoral pain by other mechanisms such as an increased sensory feedback.(15) The term ‘increased sensory feedback’ is used to depict an alteration in proprioception and muscular control.(6,38,45,85) Patients with PFP have been shown to have abnormal knee joint proprioception.(2,56) Abnormal patellar tracking may result from, or cause damage to, proprioceptive nerve fibers in the peripatellar tissues.(62) Compressive sleeves have been reported in the literature to improve knee proprioception.(6,56,64,85) Birmingham et al. studied the effect of wearing a knee sleeve during active and passive movements in open and closed kinetic chains and noticed a significant improvement of proprioception in the braced condition during angle reproduction.(6) Perla et al. found the same results with the use of an elastic knee sleeve during passive knee-extension movement. (85) It has been indicated that the proprioceptive input caused by cutaneous stimulation, as a result of taping and bracing of the knee, has an impact on muscle recruitment.(115) These data suggest that lower-extremity neuromuscular control is altered when external devices, such as a brace, are applied. Although the exact underlying mechanism behind the effect of bracing on the patellofemoral joint remains uncertain, there is some evidence that the use of braces may also be effective in the treatment of patellofemoral pain by improving knee proprioception and muscular recruitment.(6,38,45,83,85,116)

## **5. Background and aims of this dissertation.**

The first aim of this doctoral dissertation was to gain a better insight into gait-related intrinsic risk factors for the development of patellofemoral pain (Chapters 2 and 3).

Nonetheless the patellofemoral dysfunction syndrome is very frequently encountered by sports medicine practitioners, the aetiological mechanisms of this disorder remain enigmatic. The motion of the foot during walking and running is one of the potential risk factors for PFP which has been addressed in a number of studies.(12,22,26,46,66,77,86) Excessive or prolonged pronation of the foot are propounded to be risk factors for PFP (26,46,59,109), however, there is no consensus in the literature concerning this factor being a predisposing factor for the development of this disorder. A possible reason for this lack of consensus is that the studies which have examined the relation between foot motion and PFP in the past are retrospective or theoretically based. Therefore, the first purpose of this project was to prospectively identify gait-related intrinsic risk factors for patellofemoral pain.

In **chapter 2**, a prospective study was set up to examine gait-related intrinsic risk factors for patellofemoral pain in male recruits during their basic military training period. The purpose of this study was to investigate if a certain roll-over pattern of the foot during walking may predispose an individual to the development of patellofemoral pain. In **chapter 3**, a prospective investigation of gait-related risk factors was performed in recreational runners. The intention of this study was to examine if a certain static foot posture and roll-over pattern of the foot during running are predisposing factors for PFP.

The second aim of this dissertation was to investigate the correlation between the strength of the hip musculature and the frontal plane movement of the knee during a functional movement (Chapter 4).

In the recent years hip muscle weakness has been targeted as one of the possible aetiological factors for PFP.(54,72,82,111) Hip abductor and hip external rotator weakness have been hypothesized to cause excessive hip adduction and internal femoral rotation, leading to an increase in the dynamic Q-angle at the knee. However, the relationship between weakness of the hip muscles and knee kinematics has not yet been examined. Therefore in **chapter 4**, we investigated whether the strength of the muscles around the hip joint is related to the frontal plane motion of the knee during a functional lunge movement. Since the studies, which have been suggesting hip muscle weakness and presumed altered hip and knee kinematics to be a risk factor for PFP, are retrospective (54,92,111), it remains elusive if the patients in these studies demonstrated this hip muscle weakness and presumed faulty lower extremity mechanics prior to developing PFP. The goal of our study therefore was to primary search for a possible relationship between hip muscle strength and the frontal plane movement of the knee during a functional movement in an asymptomatic population.

The third aim of this dissertation was to gain a better insight in the working mechanism of the use of braces in the treatment of the patellofemoral dysfunction syndrome (Chapter 5).

Besides a pure mechanical mechanism, an alteration in proprioception has been proposed as another possible mechanism by which bracing seems to influence on the prevention and treatment of PFP.(6,38,45,83,85,115) Although it has been demonstrated that knee joint proprioception improves with the application of a knee brace, today it is still uncertain what mechanism is responsible for this improvement in knee proprioception. It has been suggested by some that improvements in proprioception as a result of wearing a brace are regulated by

reflexive pathways.(6,127) However, others have suggested that the additional somatosensory cues caused by increased cutaneous stimulation are conveyed to higher motor control centres.(58,91) Today, it remains uncertain at which level of the nervous system this alteration in proprioception is regulated. Therefore, in **chapter 5**, we investigated if there is a detectable difference in brain activity during flexion-extension movement of the knee when additional proprioceptive input is applied at the knee by the application of a knee brace.

## REFERENCES

1. Bahr R, Holme I. Risk factors for sports injuries – a methodological approach. *Br J Sports Med.* 2003; 37: 384-392.
2. Baker V, Bennel K, Stillman B, Cowan S, Crossley K. Abnormal knee joint position sense in individuals with patellofemoral pain syndrome. *J Orthop Res.* 2002; 20(2): 208-214.
3. Bennel K, Bartam S, Crossley K, et al. Outcome measures in patellofemoral pain syndrome: test retest reliability and inter-relationships. *Phys Ther in Sports.* 2000; 1: 32-41.
4. Bennet JG, Stauber WT. Evaluation and treatment of anterior knee pain using eccentric exercise. *Med Sci Sports Exerc.* 1986; 18(5): 526-530.
5. Biedert RM, Warnke K. Correlation between the Q angle and the patella position: a clinical and axial computed tomography evaluation. *Arch Orthop Trauma Surg.* 2001; 121(6): 346-349.
6. Birmingham TB, Inglis JT, Kramer JF, Vandervoort AA. Effect of a neoprene sleeve on knee joint kinesthesia: comparison of active, passive and axially loaded joint angle replication tests. *Med Sci Sports Exerc.* 2000; 32: 304-308.
7. Bizzini M, Childs JD, Piva SR, Delitto A. Systematic review of the quality of randomized controlled trials for patellofemoral pain syndrome. *J Orthop Sports Phys Ther.* 2003; 33(1): 4-20.
8. Blond L, Hansen L. Patellofemoral pain syndrome in athletes: A 5-7 year retrospective follow-up study of 250 athletes. *Acta Orthopaedica Belgica.* 1998; 64(4): 393-399.
9. Boucher JP, King MA, Lefebvre R, Pepin A. Quadriceps femoris muscle activity in PF-pain syndrome. *Am J Sports Med.* 1992; 20(5): 527-532.
10. Brechter JH, Powers CM. Patellofemoral stress during walking in persons with and without patellofemoral pain. *Med Sci Sports Exerc.* 2002; 34 (10): 1582-1593.
11. Brody LT, Thein JM. Non-operative treatment for patellofemoral pain. *J Orthop Sports Phys Ther.* 1998; 28: 336-344.
12. Callaghan MJ, Baltzopoulos V. Gait analysis in patients with anterior knee pain. *Clin Biomech.* 1994; 9: 79-84.
13. Caylor D, Fites R, Worrell TW. The relationship between quadriceps angle and anterior knee pain syndrome. *J Orthop Sports Phys Ther.* 1993; 17(1): 11-16.

14. Cerny K. Vastus medialis oblique/vastus lateralis muscle activity ratios for selected exercises in persons with and without patellofemoral pain syndrome. *Phys Ther.* 1995; 75(8): 672-683.
15. Cherf J, Paulos LE. Bracing for patellar instability. *Clin Sports Med.* 1990; 9: 813-821.
16. Cheung RTH, Ng GYF, Chen BFC. Association of footwear with patellofemoral pain syndrome in runners. *Sports Med.* 2006; 36(3): 199-205.
17. Cowan SM, Bennel KL, Crossley KM, Hodges PW, McConnell. Physical therapy alters recruitment of the vasti in patellofemoral pain syndrome. *Med Sci Sports Exerc.* 2002; 34(12): 1879-1885.
18. De Haven KE, Dolan WA, Mayer PJ. Chondromalacia patellae in athletes: clinical presentation and conservative management. *Am J Sports Med.* 1979; 7: 5-11.
19. Devereaux MD, Lachmann SM. Patellofemoral arthralgia in athletes attending a sports injury clinic. *Br J Sports Med.* 1984; 18(1): 18-21.
20. Doucette SA, Child DD. The effect of open and closed chain exercise and knee joint position on patellar tracking in lateral patellar compression syndrome. *J Orthop Sports Phys Ther.* 1996; 23: 104-110.
21. Doucette SA, Goble EM. The effect of exercise on patellar tracking in lateral patellar compression syndrome. *Am J Sports Med.* 1992; 20(4): 434-440.
22. Duffey MJ, Martin DF, Cannon DW, et al. Etiologic factors associated with anterior knee pain in distance runners. *Med Sci Sports Exerc.* 2000; 32 (11): 1825-1832.
23. Dye SF, Vaupel GL, Dye CC. Conscious neurosensory mapping of the internal structures of the human knee without intra-articular anesthesia. *Am J Sports Med.* 1998; 26: 773-777.
24. Dye SF. Therapeutic implications of a tissue homeostasis approach to patellofemoral pain. *Sports Med Arthrosc Rev.* 2001; 9: 306-311.
25. Elias DA, White LM. Imaging of patellofemoral disorders. *Clinical Radiology.* 2004; 59: 543-557.
26. Eng JJ, Pierrynowski MR. Evaluation of soft foot orthotics in the treatment of patellofemoral pain syndrome. *Phys Ther.* 1993; 73(5): 62-68.
27. Escamilla RF, Fleisig GS, Zheng N, Barrentine SW, Wilk KE, Andrews JR. Biomechanics of the knee during closed kinetic chain and open kinetic chain exercises. *Med Sci Sports Exerc.* 1998; 30(4): 556-569.

28. Fairbank J, Pynsent P, Van Poortvliet J, Phillips H. Mechanical factors in the incidence of knee pain in adolescents and young adults. *J Bone Joint Surg.* 1984; 66: 685-693.
29. Farr J. Debatable Terms. *J Orthop and Sports Phys Ther.* 1991; 14(5): 182-183.
30. Finestone A, Radin EL, Lev B, Shlamkovitch N, Wiener M, Milgrom C. Treatment of overuse patellofemoral pain. Prospective randomised controlled clinical trial in a military setting. *Clin Orthop.* 1993; 293: 208-210.
31. Fulkerson JP, Arendt A. Anterior knee pain in females. *Clin Orthop.* 2000; 372: 69-73.
32. Fulkerson JP, Hungerford DS. Disorders of the patellofemoral joint. 1990; Williams & Wilkins, Baltimore.
33. Fulkerson JP, Hungerford DS. Evaluation and rehabilitation of nonarthritic anterior knee pain. In: Disorders of the patellofemoral joint. Fulkerson JP, Hungerford DS, eds. Williams & Wilkins, Baltimore. 1990: 86-101.
34. Fulkerson JP, Shea KP. Current concepts review. Disorders of patellofemoral alignment. *J Bone Joint Surg.* 1990; 72: 1424-1429.
35. Fulkerson JP. Awareness of the retinaculum in evaluating patellofemoral pain. *Am J Sports Med.* 1982; 10(3): 147-149.
36. Fulkerson JP. Diagnosis and treatment of patients with patellofemoral pain. *Am J Sports Med.* 2002; 30(3): 447-456.
37. Fulkerson JP. The etiology of patellofemoral pain in young active patients: A prospective study. *Clin Orthop.* 1983; 179: 129-133.
38. Gilleard W, McConnell J, Parsons D. The effect of patellar taping on the onset of vastus medialis obliquus and vastus lateralis muscle activity in persons with patellofemoral pain. *Phys Ther.* 1998; 78: 25-32.
39. Goldberg B. Chronic anterior knee pain in the adolescent. *Pediatric Annals.* 1991; 20: 186-193.
40. Grabiner MD, Koh TJ, Draganich M. Neuromechanics of the PF joint. *Med Sci Sports Exerc.* 1994; 26(1): 10-21.
41. Grana WA, Kriegshauser LA. Scientific basis of extensor mechanism disorders. *Clin Sports Med.* 1985; 4: 247-257.
42. Greenwald AE, Bagley AM, France EP, Paulos LE, Greenwald RM. A biomechanical and clinical evaluation of a patellofemoral knee brace. *Clin Orthop.* 1996; 324: 187-195.
43. Grelsamer RP. Patellar malalignment. *J Bone and Joint Surg.* 2000; 82(11): 1639-1650.

44. Gross MT, Foxworth JL. The role of foot orthoses as an intervention for patellofemoral pain. *J Orthop Sports Phys Ther.* 2003; 33(11): 661-670.
45. Guling LK, Lephart SM, Stone DA, Irrgang JJ, Pincivero DM. The effect of patellar bracing on quadriceps EMG activity during isokinetic exercise. *Isokin Exerc Sci.* 1996; 6: 133-138.
46. Hamill J, Bates BT, Holt KG. Timing of lower extremity joint actions during treadmill running. *Med Sci Sports Exerc.* 1992; 24(7): 807-13.
47. Heegaard J, Leyvraz PF, Curnier A; Rakotomanana L; Huiskes R. The biomechanics of the human patella during passive knee flexion. *J Biomechanics.* 1995; 28(11): 1265-1279.
48. Hetsroni I, Finestone A, Milgrom C, Ben Sira D, Nyska M, Radeva-Petrova D, Ayalon M. A prospective biomechanical study of the association between foot pronation and the incidence of anterior knee pain among military recruits. *J Bone Joint Surg.* 2006; 88(7): 905-908.
49. Hodges PW, Richardson CA. The influence of isometric hip adduction on quadriceps femoris activity. *Scand J Rehabil Med.* 1993; 25(2): 57-62.
50. Hodgson Phillips L. Sports injury incidence. *Br J Sports Med.* 2000; 34: 133-136.
51. Holmes WS, Clancy WG. Clinical classification of patellofemoral pain and dysfunction. *JOSPT.* 1998; 28(5): 299-306.
52. Insall J, Falvo KA, Wise DW. Chondromalacia patellae. *J Bone Joint Surg.* 1976 ; 58(1) ; 1-8.
53. International Patellofemoral Study Group. Patellofemoral semantics: The Tower of Babel. *Am J Knee Surg.* 1997; 10: 92-95.
54. Ireland ML, Willson JD, Ballantyne BT, Davis IM. Hip strength in females with and without patellofemoral pain. *J Orthop Sports Phys Ther* 2003; 33(11): 671-76.
55. Jerosch J, Prymka M. Knee joint proprioception in normal volunteers and patients with anterior cruciate ligament tears, taking special account of the effect of a knee bandage. *Arch Orthop trauma Surg.* 1996; 115: 162-166.
56. Jerosch J, Schmidt K, Prymka M. Proprioceptive capacities of patients with retropatellar knee pain with special reference to effectiveness of an elastic knee bandage. *Unfallchirurg.* 1997; 100(9): 719-723.
57. Johnston LB, Gross MT. Effects of foot orthoses on quality of life for individuals with patellofemoral pain syndrome. *J Orthop Sports Phys Ther.* 2004; 34(8): 440-447.
58. Kaminski T, Perrin DH, 1996. Effect of prophylactic knee bracing on balance and joint position sense. *J Atl Training.* 31, 2, 131-136.



59. Klingman RE, Liaos SM, Hardin KM. The effect of subtalar joint posting on patellar glide position in subjects with excessive rearfoot pronation. *J Orthop Sports Phys Ther.* 1997; 25(3): 185-191.
60. Klingman RE. Foot pronation and patellofemoral jointfunction. *J Orthop Sports Phys Ther.* 1999; 29(7): 421.
61. Kramer PG. Patella malalignment syndrome/ Rationale to reduce excessive lateral pressure. *J Orthop Sports Phys Ther.* 1986; 8: 301-309.
62. LaBella C. Patellofemoral pain syndrome: evaluation and treatment. *Prim Care Clin Office Pract.* 2004; 31: 977-1003.
63. Lam PL, Gyf N. Activation of the quadriceps muscle during semisquatting with different hip and knee positions in patients with anterior knee pain. *Am J Phys Med Rehabil.* 2001; 80(11): 804-808.
64. Lephart S, Kocher MS, Fu FH, Borsa PA, Harner CD. Proprioception following anterior cruciate ligament reconstruction. *J Sport Rehabil.* 1992; 1: 188-199.
65. Leslie IJ, Bentley G. Arthroscopy in the diagnosis of chondromalacia patellae. *Ann Rheum Dis.* 1978; 37: 540-547.
66. Levinger P, Gilleard W. An evaluation of the rearfoot posture in individuals with patellofemoral pain syndrome. *J Sports Sci and Med.* 2004; 3: 8-14.
67. Livingston LA, Spaulding SJ. OPTOTRAK measurement of the Quadriceps angle using standardized foot positions. *Journal of Athletic Training.* 2002; 37(3): 252-55.
68. Livingston LA. The quadriceps angle: a review of the literature. *J Orthop Sports Phys Ther.* 1998; 28: 105-109.
69. Llopis E, Padron M. Anterior knee pain. *European Journal of Radiology.* 2007; 62: 27-43.
70. Lun V, Meeuwisse W, Stergiou P, Stefanyshyn D. Relation between running injury and static lower limb alignment in recreational runners. *Br J Sports Med.* 2004; 38: 576-580.
71. Mariani PP, Caruso I. An electromyographic investigation of subluxation of the patella. *J Bone Joint Surg (Br).* 1989; 61: 169-171.
72. Mascal CL, Landel R, Powers C. Management of patellofemoral pain targeting hip, pelvis and trunk muscle function: 2 case reports. *J Orthop Sports Phys Ther* 2003; 33(11): 647-60.
73. McConnell J. The management of chondromalacia patellae: a long term solution. *Australian Journal of Physiotherapy.* 1986; 32: 215-223.

74. McNally EG, Ostlere SJ, Pal C, Phillips A, Reid H, Dodd C. Assessment of patellar maltracking using combined static and dynamic MRI. *Eur Radiol.* 2000; 10: 1051-1055.
75. McNally EG. Imaging assessment of anterior knee pain and patellar maltracking. *Skeletal Radiol.* 2001; 30: 484-495.
76. Meeuwisse, WH. Assessing causation in sport injury: A multifactorial model. *Clin J Sports Med.* 1994; 4: 166-170.
77. Messier SP, Davis SE, Curl WW, Lowerly RB, Pack RJ. Etiologic factors associated with patellofemoral pain in runners. *Med Sci Sports Exerc.* 1991; 23 (9): 1008-1015.
78. Milgrom C, Finestone A, Eldad A, Shlamkovitch N. Patellofemoral pain caused by overactivity. A prospective study of risk factors in infantry recruits. *J Bone Joint Surg.* 1991; 73: 1041-1043.
79. Milgrom C, Finestone A, Shlamkovitch N, Giladi, Radin E. Anterior knee pain caused by overactivity: a long term prospective follow up. *Clin Orthop.* 1996; 331: 256-260.
80. Miller JP, Sedory D, Croce RV. Vastus medialis obliquus and vastus lateralis activity in patients with and without patellofemoral pain syndrome. *J Sport Rehab.* 1997; 6: 1-10.
81. Muhle C, Brinkmann G, Skaf A, Heller M, Resnick D. Effect of a patellar realignment brace on patients with patellar subluxation and dislocation. *Am J Sports Med.* 1999; 27: 350-353.
82. Niemuth PE, Johnson RJ, Myers MJ, Thieman TJ. Hip muscle weakness and overuse injuries in recreational runners. *Clin J Sport Med* 2005; 15(1); 14-21.
83. Osternig LR, Robertson RN. Effects of prophylactic knee bracing on lower extremity position and muscle activation during running. *Am J Sports Med.* 1993; 21: 733-737.
84. Palmitier RA, An KN, Scott SG, Chao EY. Kinetic chain exercise in knee rehabilitation. *Sports Med.* 1991; 11: 402-413.
85. Perlau R, Frank C, Fick G. The effect of elastic bandages on human knee proprioception in the uninjured population. *Am J Sports Med.* 1995; 23: 251-255.
86. Powers CM, Chen PY, Reischl SF, Perry J. Comparison of foot pronation and lower extremity rotation in persons with and without patellofemoral pain. *Foot ankle Int.* 2002; 23 (7): 634-40.
87. Powers CM, Landel R, Perry J. Timing and intensity of vastus muscle activity during functional activities in subjects with and without patellofemoral pain. *Phys Ther.* 1996; 76: 946-955.

88. Powers CM. The influence of altered lower-extremity kinematics on patellofemoral joint dysfunction: A theoretical Perspective. *J Orthop Sports Phys Ther.* 2003; 33: 639-646.
89. Razeghi M, Batt ME. Biomechanical analysis of the effect of orthotic shoe inserts: A review of the literature. *Sports Med.* 2000; 29(6): 425-438.
90. Reider B, Marshall JL, Warren RF. Clinical characteristics of patellar disorders in young athletes. *Am J Sports Med.* 1981; 9: 270-274.
91. Riemann BL, Lephart SM, 2002. The sensorimotor system, Part II: The role of proprioception in motor control and functional joint stability. *Journal of Athletic Training.* 37, 1, 80-84.
92. Robinson RL, Nee RJ. Analysis of hip strength in females seeking physical therapy treatment for unilateral patellofemoral pain syndrome. *J Orthop Sports Phys Ther.* 2007; 37(5): 232-238.
93. Royle SG, Noble J, Davies DR. The significance of chondromalacic changes of the patella. *Arthroscopy.* 1991; 7: 158-160.
94. Rutherford OM. Muscular coordination and strength training. Implications for injury rehabilitation. *Sports Med.* 1988; 5: 196-202.
95. Salsich GB, Ward SR, Terk MR, Powers CM. In vivo assessment of patellofemoral joint contact area in individuals who are pain free. *Clin Orthop.* 2003; 417: 277-284.
96. Sanchis-alfonso V, Prat-Pastor J, Atienza-Vicente CM, Puig-ABBS C, Comin-Clavijo M. Biomechanical bases for anterior knee pain and patellar instability in the young patient. In: *Anterior knee pain and patellar instability.* Vicente Sanchis-Alfonso, ed. Springer, London. 2006: 147-166.
97. Schulties SS, Francis RS, Fisher AG, Van De Graaff KM. Does the Q angle reflect the force on the patella in the frontal plane? *Physical Therapy.* 1995; 75(1): 30-36.
98. Shellock FG, Mink JH, Deutsch AL, Fox J, Molnar T. Effect of a newly-designed patellar realignment brace on patellofemoral relationships. *Med Sci Sports Exerc.* 1995; 27: 469-472.
99. Skalley TC, Terry GC, Temtge RA. The quantitative measurement of normal passive medial and lateral patellar motion limits. *Am J Sports Med.* 1993; 21(5): 728-732.
100. Souza DR, Gross MT. Comparison of VMO:VL muscle integrated EMG ratios between healthy subjects and patients with PF-pain. *Phys Ther.* 1990; 71(4): 310-320.
101. Stefanyshyn DJ, Stergiou P, Lun VMY. Knee angular impulse as a predictor of patellofemoral pain in runners. *Am J Sports Med.* 2006; 34(11): 1844-1851.

102. Steinkamp LA, Dillingham MF, Markel MD, Hill JA, Kaufman KR. Biomechanical considerations in patellofemoral joint rehabilitation. *Am J Sports Med.* 1993; 21: 438-444.
103. Stiene HA, Brosky T, Reinking MF, Nyland J, Mason MB. A comparison of closed kinetic chain and isokinetic joint isolation exercise in patients with patellofemoral dysfunction. *JOSPT.* 1996; 24: 136-421.
104. Subotnik S. The foot and sports medicine. *J Orthop Sports Phys Ther.* 1980; 2(2): 53-54.
105. Taimela S. Intrinsic risk factors and athletic injuries. *Sports Med.* 1990; 9: 205-215.
106. Tang SF, Chen CK, Hsu R, Chou SW, Hong WH, Lew HL. Vastus medialis obliquus and vastus lateralis activity in open and closed kinetic chain exercises in patients with patellofemoral pain syndrome: An electromyographic study. *Arch Phys Med Rehab.* 2001; 82: 1441-1445.
107. Thomée P, Thomée R, Karlsson J. Patellofemoral pain syndrome : pain, coping strategies and degree of well-being. *Scand J Med Sci Sports.* 2002; 12: 276-281.
108. Thomée R, Renstrom P, Karlsson J. Patellofemoral pain syndrome in young women. *Scand J Med Sci Sports.* 1995; 5: 245-251.
109. Tiberio D. The effect of excessive subtalar joint pronation on patellofemoral mechanics: A theoretical model. *J Orthop Sports Phys Ther.* 1987; 9: 160-165.
110. Tria AJ, Palmubo RC, Alicea JA. Conservative care for patellofemoral pain. *Orthop Clin North Am.* 1992; 23: 545-554.
111. Tyler TF, Nicholas SJ, Mullaney MJ, McHugh MP. The role of hip muscle function in the treatment of patellofemoral pain syndrome. *Am J Sports Med.* 2006; 35: 1-7.
112. van Gent RN, Siem D, van Middelkoop M, Van Os, Bierma-Zeinstra SMA, Koes BW. Incidence and determinants of lower extremity running injuries in long distance runners: a systematic review. *Br J Sports Med.* 2007; 41: 469-480.
113. Van Mechelen W, Hlobil H, Kemper HC. Incidence, severity, aetiology and prevention of sports injuries. A review of concepts. *Sports Med.* 1992; 14:82-99.
114. Van Mechelen W. Running injuries. A review of the epidemiological literature. *Sports Med.* 1992; 14:320-35.
115. Van Tiggelen D, Coorevits P, Witvrouw E. The effects of a neoprene knee sleeve on subjects with a poor versus good joint position sense subjected to an isokinetic fatigue protocol. *Clin J Sport Med.* 2008; 18 (3): 259-265.

116. Van Tiggelen D, Coorevits P, Witvrouw E. The use of a neoprene knee sleeve to compensate the deficit in knee joint position sense caused by muscle fatigue. *Scand J Med Sci Sports*. 2008; 18: 62-66.
117. Voight M, Wieder DL. Comparative reflex response times of VMO and VL in normal subjects and in subjects with extensor mechanism dysfunction. *Am J Sports Med*. 1991; 19(2): 131-136.
118. Werner S. An evaluation of knee extensor and knee flexor torques and EMG's in patients with patellofemoral pain syndrome in comparison with matched controls. *Knee Surg Sports Traumatol Arthrosc*. 1995; 3: 89-94.
119. Werner S. Conservative treatment of athletes with anterior knee pain. Science: Classical and new ideas. In: *Anterior knee pain and patellar instability*. Vicente Sanchis-Alfonso, ed. Springer, London. 2006: 147-166.
120. Wild JJ, Franklin TD, Woods GW. Patellar pain and quadriceps rehabilitation. *Am J Sports Med*. 1982; 10: 12-15.
121. Witvrouw E, Cambier D, Danneels L, Bellemans J, Almqvist F, Verdonk R. The effect of exercise regimes on reflex response time of the vasti muscles in patients with anterior knee pain: a prospective randomized intervention study. *Scand J Med Sci Sports*. 2003; 13(4): 251-258.
122. Witvrouw E, Lysens R, Bellemans J, Cambier D, Vanderstraeten G. Intrinsic risk factors for the development of anterior knee pain in an athletic population: A two-year prospective study. *Am J Sports med*. 2000; 28 (4): 480-489.
123. Witvrouw E, Lysens R, Bellemans J, Peers K, Vanderstraeten G. Open versus closed kinetic chain exercises for patellofemoral pain: A prospective, randomized study. *Am J Sports Med*. 2000; 28(5): 687-694.
124. Witvrouw E, Sneyers C, Lysens R, Victor J, Bellemans J. Reflex response times of vastus medialis oblique and vastus lateralis in normal subjects and in subjects with patellofemoral pain. *JOSPT*. 1996; 24(3): 160-165.
125. Witvrouw E, Werner S, Mikkelsen C, Van Tiggelen D, Vanden Berghe L, Cerulli G. Clinical classification of patellofemoral pain syndrome: guidelines for non-operative treatment. *Knee Surg Sports Traumatol Arthrosc*. 2005; 13: 122-130.
126. Woodall W, Welsh J. A Biomechanical basis for rehabilitation programs involving the patellofemoral joint. *J Orthop Sports Phys Ther*. 1990; 11: 535-542.
127. You SH, Granata KP, Bunker LK. Effects of circumferential ankle pressure on ankle proprioception, stiffness and postural stability: a preliminary investigation. *J Orthop Sports Phys Ther*. 2004; 34: 449-460.

128. Sanchis-Alfonso V, Puig-Abbs C, Martinez-Sanjuan V. Evaluation of the patient with anterior knee pain and patellar instability. In: Anterior knee pain and patellar instability. Vicente Sanchis-Alfonso, ed. Springer, London. 2006: 93-113.
129. Schutzer SF, Ramsby GR, Fulkerson JP. Computed tomographic classification of patellofemoral pain patients. *Orthopedic Clinics of North America*. 1986; 17 (2): 235-247.
130. Ward SR, Powers CM. The influence of patella alta on patellofemoral joint stress during normal and fast walking. *Clin Biomech*. 2004; 19: 1040-1047.
131. Davies AP, Costa ML, Shepstone L, Glasgow MM, Donell S, Donell ST. The sulcus angle and malalignment of the extensor mechanism of the knee. *J Bone and Joint Surg Br*. 2000; 82 (8): 1162-1166.
132. Schoettle PB, Zanetti M, Seifert B, Pfirrmann CWA, Fucentese SF, Romero J. The tibial tuberosity-trochlear groove distance; a comparative study between CT and MRI scanning. *The Knee*. 2006; 13: 26-31.
133. Teitge RA, Torga-Spak R. Skeletal malalignment and anterior knee pain: Rationale, Diagnosis, and Management. In: Anterior knee pain and patellar instability. Vicente Sanchis-Alfonso, ed. Springer, London. 2006: 185-199.
134. Goutallier D, Bernageau J, Lecudonnec B. Mesure de l'écart tubérosité tibiale antérieure-gorge de la trochlée (TA-GT). *Rev Chir Orthop*. 1978; 64 : 423-428.
135. Witonski D, Goraj B. Patellar motion analysed by kinematic and dynamic axial magnetic resonance imaging in patients with anterior knee pain syndrome. *Arch Orthop Trauma Surg*. 1999; 119: 46-49.
136. Laprade J, Culham E, Brouwer B. Comparison of five isometric exercises in the recruitment of the vastus medialis oblique in persons with and without patellofemoral pain syndrome. *JOSPT*. 1998; 27 (3): 197-204.
137. Mirzabeigi E, Jordan C, Gronley J, Rockowitz NL, Perry J. Isolation of the vastus medialis oblique muscle during exercise. *Am J Sports Med*. 1999; 27 (1): 50-53.
138. Werner S, Arvidsson H, Arvidsson I, Eriksson E. Electrical stimulation of vastus medialis and stretching of lateral thigh muscles in patients with patellofemoral symptoms. *Knee Surg Sports Traumatol Arthrosc*. 1993; 1 (2): 85-92.

## **CHAPTER 2**

### **A prospective study on gait related intrinsic risk factors for patellofemoral pain**

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## Abstract

**Objective:** To prospectively determine gait related risk factors for patellofemoral pain.

**Design:** A prospective cohort study.

**Setting:** Male and female recruits of the Belgian Royal Military Academy during a 6 week basic military training period.

**Participants:** 84 officer cadets (65 men, 19 women), who entered the Military Academy and were without a history of any knee or lower leg complaints participated in the study.

**Interventions:** Before the start of the 6 week basic military training period plantar pressure measurements during walking were performed. During the basic military training period, patellofemoral complaints were diagnosed and registered by a Sports Medicine Physician.

**Main Outcome Measurements:** Plantar pressure measurements during walking were performed using a footscan pressure plate (RsScan International).

**Results:** During the six week training period 36 subjects developed patellofemoral pain (25 male and 11 female). Logistic regression analysis revealed that subjects who developed patellofemoral pain had a significantly more laterally directed pressure distribution at initial contact of the foot, a significantly shorter time to maximal pressure on the fourth metatarsal and a significantly slower maximal velocity of the change in latero-medial direction of the centre of pressure during the forefoot contact phase.

**Conclusions:** Our findings suggest that the feet of the persons who developed anterior knee pain have a heel strike in a less pronated position and rollover more on the lateral side compared with the control group. The results of this study can be considered as valuable in identifying persons at risk for patellofemoral pain.

**Key words:** knee; anterior knee pain; foot rollover; plantar pressure



## INTRODUCTION

Patellofemoral pain (PFP) is one of the most common disorders involving the knee seen by sports medicine practitioners<sup>1, 2, 3, 4, 5, 6, 7</sup>. The literature describes an incidence as high as one in four in the general population and even higher among athletes<sup>8, 9, 10</sup>. Therefore, patellofemoral pain represents a major problem in a wide range of individuals, particularly in physically active populations such as adolescents and young adults, athletes<sup>2, 5, 6, 10, 11</sup> and military personnel<sup>12</sup>. Despite its high incidence there is a lack of consensus concerning the aetiological mechanisms of this disorder. Focusing on the joint itself, numerous risk factors are suggested including patellofemoral malalignment, soft periarticular tissue imbalances, quadriceps muscle weakness, vasti muscle imbalance and bony abnormalities<sup>7, 10, 13</sup>. However, it has been stated in the literature that the causes of patellofemoral pain are multifactorial<sup>14</sup>.

It has been recognized that patellofemoral joint mechanics may be influenced by segmental interactions of the lower extremity<sup>6</sup>. Abnormal lower extremity kinematics has been commonly cited as a possible predisposing factor for PFP<sup>7</sup>.

Therefore, a number of studies have investigated gait related risk factors as potential aetiological factors for patellofemoral pain<sup>2, 4, 5, 6, 7, 13, 15, 16, 17, 18</sup>. Abnormal foot pronation and subsequent lower extremity rotation have been focused, with excessive or prolonged foot pronation propounded to be predisposing for PFP<sup>16, 18</sup>.

In the literature, however, there is no consensus concerning abnormal rearfoot pronation being a risk factor for the development of anterior knee pain. This suggests that abnormal pronation is not a universal finding in patients suffering from patellofemoral pain and, as has been stated by Powers et al.<sup>7</sup>, care must be taken in attributing the cause of PFP symptoms to abnormal pronation.

One of the reasons for this lack of consensus regarding this issue might be that the studies which have investigated the relationship between rearfoot movement and PFP are all retrospective<sup>2, 4, 5, 7, 13, 16, 17</sup> or based on a theoretical model<sup>6, 15, 18</sup>. However to predict injury, studies must measure potential risk factors in subjects before the occurrence of injury<sup>19</sup>. Cross-sectional studies can associate potential risk factors with certain injuries but longitudinal prospective studies can investigate cause and effect relationships. To date, to our knowledge, no prospective studies exist which have investigated the relationship between the rollover pattern of the foot during gait and the development of anterior knee pain. Therefore, the purpose of this study was to prospectively determine gait related risk factors for patellofemoral pain.

## **METHODS**

### **Subjects**

Eighty-four officer cadets (65 men, 19 women) who had no history of any knee or lower leg complaints were prospectively examined. These persons were recruited from the total of 105 officer cadets of the Belgian Royal Military Academy, who entered the Academy in August 2006. Before testing, all cadets visited the same sports medicine physician for a comprehensive injury history. Twenty-one recruits, who had a history of a surgical procedure involving the knee, lower leg, ankle or foot or a history of an injury to the knee, lower leg, ankle or foot within six months before the start of the study were excluded from the study. The average age of the subjects, included in the study, was 19 years ( $SD \pm 1.54$ ). The cadets had an average height of 177.9 cm ( $SD \pm 7.78$ ) and an average weight of 67.5 kg ( $SD \pm 7.92$ ). The aim of the study was explained to each subject and they all signed an informed consent. For the subjects under the age of 18, parental consent was obtained. The study was approved by the Ethical Commission of Belgian Defense.

### **Evaluation**

The gait pattern of the subjects was examined before the start of a 6 week basic military training period using a footscan pressure plate (RsScan International). This device has revealed deviant gait characteristics in previous prospective studies<sup>20</sup>.

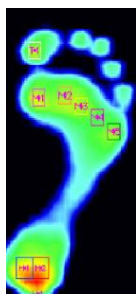
### **Plantar pressure measurements**

Before the start of the training period all cadets underwent plantar pressure measurements during barefoot walking. Plantar pressure measurements were performed during walking because this activity represents the major activity the cadets performed during the basic military training period. By walking barefoot, discrepancies due to unusual footwear were controlled. Clarke et al.<sup>21</sup> stated that in running analysis barefoot is an accepted baseline state and it has been shown that gait related risk factors for exercise related lower leg pain are more distinct in the barefoot condition compared to the shod condition<sup>22</sup>. A footscan pressure plate (RsScan International, 0,5m x 0,4m, 295 Hz) was flush mounted in the middle of a 15 m long walkway. The walkway was covered by a thin rubber mat so that the pressure plate would not be visible to the subjects.

The subjects were asked to walk at a self chosen comfortable moderate velocity along the walkway. All subjects were allowed to familiarize themselves with the procedures before data collection. Three valid left and right stance phases were measured. A trial was considered to be valid when there was a heel strike pattern, no adjustment in step length or frequency to aim on the pressure plate. De Cock et al.<sup>23</sup> found the temporal plantar pressure variables measured with the footscan<sup>®</sup> pressure plate to be reliable (ICC > 0.75).

## Data analysis

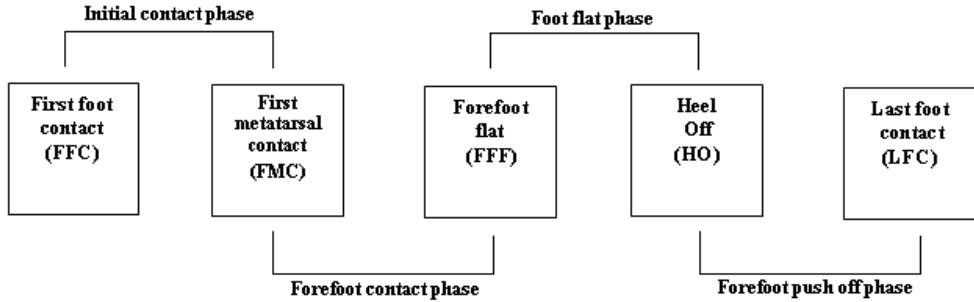
For each trial, eight anatomical pressure areas were manually identified, based on the peak pressure footprint (figure 1). These eight anatomical areas were defined as medial heel (H1), lateral heel (H2), metatarsal heads I – V (M1, M, M3, M4, M5) and the hallux (T1).



**Figure 1:** Location of the eight anatomical pressure areas. (RsScan International)

Temporal data (time to peak pressure, instants on which the regions make contact and instants on which the regions end contact), peak pressure data and absolute impulses (mean pressure x loaded contact time) and relative impulses (absolute impulse x 100 / sum of all impulses) were calculated for all eight regions. The total foot contact time and five distinct instants of foot rollover were determined for each trial. These five instants of foot rollover were: first foot contact (FFC), first metatarsal contact (FMC), forefoot flat (FFF), heel off (HO) and last foot contact (LFC). FFC was defined as the instant the foot made first contact with the pressure plate. FMC was defined as the moment when one of the metatarsal heads contacted the pressure plate. FFF was defined as the first instant all metatarsal heads made contact with the pressure plate. HO was defined as the instant the heel region ended contact with the pressure plate. LFC was defined as the last contact of the foot on the plate. Based on these instants, total foot contact could be divided into four phases: initial contact phase (ICP = FFC

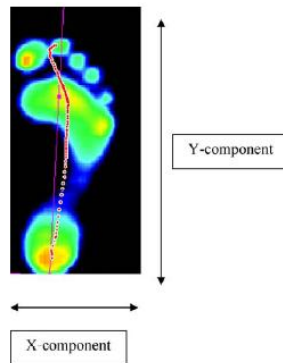
to FMC), forefoot contact phase (FFCP = FMC to FFF), foot flat phase (FFP = FFF to HO) and forefoot push off phase (FFPOP = HO to LFC) (figure 2).



**Figure 2:** Five distinct instants and four phases of foot rollover

A medio-lateral pressure ratio  $[(M1 + M2 + H1)/(M4 + M5 + H2)]$  was calculated at the five instants of foot contact. This ratio describes the pressure distribution in the foot at the five instants of foot contact. Excursion ranges of this ratio were calculated over the four phases (ICP, FFCP, FFP, FFPOP).

The X-component (medio-lateral) and Y-component (anterior- posterior) of the centre of pressure (COP) scaled to the foot width and foot length, respectively, were analyzed (figure 3). The positioning and displacements of the components were calculated at the five instants and in the four phases.



**Figure 3:** The X-component (medio-lateral) and Y-component (anterior-posterior) of the COP. (RsScan International)

The mean of all kinetic data was taken from the three trials. De Cock et al. have shown that the mean of three trials is sufficient for analysis<sup>23, 24</sup>. The plantar pressure variables are listed in table 1.

**Table 1:** List of plantar pressure variables

Plantar pressure variables	
Temporal data	Time of first lateral heel contact Time of first medial heel contact Time of contact on metatarsal heads 1, 2, 3, 4 and 5 Time of first hallux contact Time of ending lateral heel contact Time of ending medial heel contact Time of ending contact on metatarsal heads 1, 2, 3, 4 and 5 Time of ending hallux contact Time to peak pressure underneath the lateral heel Time to peak pressure underneath the medial heel Time to peak pressure underneath metatarsal heads 1, 2, 3, 4 and 5 Time to peak pressure underneath the hallux
Peak pressure data	Peak pressure underneath the lateral heel Peak pressure underneath the medial heel Peak pressure underneath metatarsal heads 1, 2, 3, 4 and 5 Peak pressure underneath the hallux Absolute impuls (mean pressure x loaded contact time) underneath the lateral heel Absolute impuls (mean pressure x loaded contact time) underneath the medial heel Absolute impuls (mean pressure x loaded contact time) underneath metatarsal heads 1, 2, 3, 4 and 5 Absolute impuls (mean pressure x loaded contact time) underneath the hallux Relative impuls (absolute impuls x 100/sum of all impulses) underneath the lateral heel Relative impuls (absolute impuls x 100/sum of all impulses) underneath the medial heel Relative impuls (absolute impuls x 100/sum of all impulses) underneath metatarsal heads 1, 2, 3, 4 and 5 Relative impuls (absolute impuls x 100/sum of all impulses) underneath the hallux
Medio-lateral pressure distribution in the foot	Medio-lateral pressure ratio $[(M1+M2+H1)/(M4+M5+H2)]$ at first foot contact Medio-lateral pressure ratio $[(M1+M2+H1)/(M4+M5+H2)]$ at first metatarsal contact Medio-lateral pressure ratio $[(M1+M2+H1)/(M4+M5+H2)]$ at forefoot flat Medio-lateral pressure ratio $[(M1+M2+H1)/(M4+M5+H2)]$ at heel off Medio-lateral pressure ratio $[(M1+M2+H1)/(M4+M5+H2)]$ at last foot contact Medio-lateral pressure ratio $[(M1+M2+H1)/(M4+M5+H2)]$ during initial contact phase Medio-lateral pressure ratio $[(M1+M2+H1)/(M4+M5+H2)]$ during forefoot contact phase Medio-lateral pressure ratio $[(M1+M2+H1)/(M4+M5+H2)]$ during foot flat phase Medio-lateral pressure ratio $[(M1+M2+H1)/(M4+M5+H2)]$ during forefoot push off phase
Displacements of the centre of pressure (COP)	Medio-lateral displacement COP at first foot contact Medio-lateral displacement COP at first metatarsal contact Medio-lateral displacement COP at forefoot flat Medio-lateral displacement COP at heel off Medio-lateral displacement COP at last foot contact Medio-lateral displacement COP during initial contact phase Medio-lateral displacement COP during forefoot contact phase Medio-lateral displacement COP during foot flat phase Medio-lateral displacement COP during forefoot push off phase Velocity of medio-lateral displacement COP at first foot contact Velocity of medio-lateral displacement COP at first metatarsal contact Velocity of medio-lateral displacement COP at forefoot flat Velocity of medio-lateral displacement COP at heel off Velocity of medio-lateral displacement COP at last foot contact Velocity of medio-lateral displacement COP during initial contact phase Velocity of medio-lateral displacement COP during forefoot contact phase Velocity of medio-lateral displacement COP during foot flat phase Velocity of medio-lateral displacement COP during forefoot push off phase Maximal velocity of medio-lateral displacement COP during initial contact phase Maximal velocity of medio-lateral displacement COP during forefoot contact phase Maximal velocity of medio-lateral displacement COP during foot flat phase Maximal velocity of medio-lateral displacement COP during forefoot push off phase

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Anterior-posterior displacement COP at first foot contact  
 Anterior-posterior displacement COP at first metatarsal contact  
 Anterior-posterior displacement COP at forefoot flat  
 Anterior-posterior displacement COP at heel off  
 Anterior-posterior displacement COP at last foot contact  
 Anterior-posterior displacement COP during initial contact phase  
 Anterior-posterior displacement COP during forefoot contact phase  
 Anterior-posterior displacement COP during foot flat phase  
 Anterior-posterior displacement COP during forefoot push off phase  
 Velocity of anterior-posterior displacement COP at first foot contact  
 Velocity of anterior-posterior displacement COP at first metatarsal contact  
 Velocity of anterior-posterior displacement COP at forefoot flat  
 Velocity of anterior-posterior displacement COP at heel off  
 Velocity of anterior-posterior displacement COP at last foot contact  
 Velocity of anterior-posterior displacement COP during initial contact phase  
 Velocity of anterior-posterior displacement COP during forefoot contact phase  
 Velocity of anterior-posterior displacement COP during foot flat phase  
 Velocity of anterior-posterior displacement COP during forefoot push off phase  
 Maximal velocity of anterior-posterior displacement COP during initial contact phase  
 Maximal velocity of anterior-posterior displacement COP during forefoot contact phase  
 Maximal velocity of anterior-posterior displacement COP during foot flat phase  
 Maximal velocity of anterior-posterior displacement COP during forefoot push off phase

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## **Basic military training**

All of the 84 officer cadets, included in this study, followed the same six week basic military training. The training mainly consisted of roadwork and marching with backpacks (35%), military tactical exercises (25%), drills (10%), running (5%), shooting (5%) and some theoretical classes (20%). Because all recruits followed the same training program with the same equipment, environmental conditions and daily schedule, the extrinsic contributing factors that could affect the incidence of patellofemoral pain were kept mainly under control.

## **Registration of injuries**

During the six week basic military training, patellofemoral complaints were diagnosed and registered by the same Sports Medicine Physician who was present at the training camp during this period. The sports medicine physician who made the injury diagnosis was blinded to the results of the plantar pressure measurements. The complaints were listed on a registration form containing information about the injury. To be considered as a patellofemoral pain patient, subjects had to have a characteristic history and symptoms of patellofemoral pain syndrome and exhibit two of the following clinical criteria on assessment: Pain on direct compression of the patella against the femoral condyles with the knee in full extension, tenderness of the posterior surface of the patella on palpation, pain on resisted knee extension, pain with isometric quadriceps muscle contraction against suprapatellar resistance

with the knee in 15° of flexion. In addition, subjects had to have negative findings in the examination of knee ligaments, menisci, bursae, synovial plicae, Hoffa's fat pad, iliotibial band, the hamstrings, quadriceps and patellar tendons and their insertions. Previous studies indicate that these criteria are sensitive and specific for diagnosing patellofemoral pain<sup>10, 25, 26</sup>.

### **Statistical analysis**

During the six week training period 36 subjects (25 male and 11 female) developed patellofemoral pain. Of these 36 subjects, 17 developed bilateral complaints. Consequently, the patellofemoral pain group comprised 53 knees. The control group consisted of both knees of the 48 uninjured subjects of the total group of 84 officer cadets. Statistical analysis was performed with SPSS for windows version 12.0 (SPSS Inc, Chicago, Ill). Prior to the statistical analysis, data from both legs of the same subject were collapsed into a single measure by taking the average of the two data points in order to make an adjustment for the correlation between the two legs of the same person.<sup>27</sup> A binary logistic regression analysis<sup>28</sup> was performed to identify the gait related intrinsic risk factors for patellofemoral pain. Firstly, in order to reduce the number of variables, student's *t*-tests (if normal data distribution) or Mann-Whitney U-tests (if no normal data distribution was obtained) were undertaken for examining possible differences between the patellofemoral pain group and the control group for each of the test variables. All variables showing a P-value < 0,1 in the univariate analysis were entered separately into the logistic regression analysis. Statistical significance was accepted at the level of  $\alpha \leq 0.05$ .

### **RESULTS**

There were no significant differences between the group of cadets who developed patellofemoral pain and those who did not with respect to their average age, height and weight. The anthropometric data of the group of cadets who developed PFP and the control group are presented in table 2.

**Table 2:** Mean, standard deviation and p-value for the t-test of the age, height and weight of the group of recruits with PFP and the control group

	Mean PFP group	SD PFP group	Mean control group	SD control group	Significance t-test
Age (years)	19.06	1.91	19.02	1.21	0.917
Height (cm)	175.94	7.54	179.28	7.73	0.054
Weight (kg)	67.60	8.41	67.40	7.63	0.913

( $\alpha = 0.05$ )

Logistic regression analysis revealed that the time to maximal pressure on the fourth metatarsal was significantly shorter ( $P = 0.009$ ) in the subjects who developed patellofemoral pain. Logistic regression did not identify any significant differences for the other temporal pressure data (table 3).

**Table 3:** Mean, standard deviation and p-value for the t-test or Mann-whitney U-test (MWU-test) ( $\alpha = 0.1$ ) and logistic regression analysis ( $\alpha = 0.05$ ) of the time to first contact, time to end of contact and time to maximal pressure on the eight anatomical areas.

	Mean control group	SD control group	Mean PFP group	SD PFP group	Significance t-test MWU-test	Significance Logistic regression
Time to contact T1 (s)	0.269	0.085	0.266	0.094	0.734	/
Time to contact M1 (s)	0.110	0.039	0.102	0.025	0.481	/
Time to contact M2 (s)	0.078	0.021	0.078	0.018	0.919	/
Time to contact M3 (s)	0.063	0.017	0.065	0.017	0.567	/
Time to contact M4 (s)	0.054	0.017	0.060	0.018	0.087 <sup>+</sup>	0.461
Time to contact M5 (s)	0.063	0.030	0.066	0.026	0.310	/
Time to contact H1 (s)	0.000	0.000	0.000	0.000	1.000	/
Time to contact H2 (s)	0.000	0.000	0.000	0.000	1.000	/
End contact T1 (s)	0.627	0.041	0.623	0.059	0.966	/
End contact M1 (s)	0.621	0.044	0.629	0.051	0.328	/
End contact M2 (s)	0.623	0.041	0.629	0.051	0.412	/
End contact M3 (s)	0.625	0.041	0.629	0.051	0.571	/
End contact M4 (s)	0.621	0.040	0.623	0.049	0.781	/
End contact M5 (s)	0.591	0.043	0.587	0.053	0.643	/
End contact H1 (s)	0.373	0.068	0.369	0.064	0.801	/
End contact H2 (s)	0.363	0.067	0.356	0.062	0.677	/
tPmax T1 (s)	0.570	0.042	0.568	0.047	0.918	/
tPmax M1 (s)	0.490	0.051	0.484	0.065	0.520	/
tPmax M2 (s)	0.523	0.045	0.521	0.043	0.779	/
tPmax M3 (s)	0.510	0.044	0.500	0.064	0.278	/
tPmax M4 (s)	0.463	0.059	0.433	0.079	0.033 <sup>+</sup>	0.009*
tPmax M5 (s)	0.402	0.077	0.380	0.086	0.268	/
tPmax H1 (s)	0.133	0.027	0.136	0.024	0.344	/
tPmax H2 (s)	0.115	0.037	0.109	0.044	0.900	/

(PFP= patellofemoral pain group, tPmax= time to max pressure, s = seconds, \* $p \leq 0.05$ , <sup>+</sup>  $p \leq 0.1$ ).

For the medio-lateral pressure ratio logistic regression showed that in the PFP group pressure distribution was significantly ( $P = 0.003$ ) more laterally directed at initial contact of the foot



(first foot contact). Mean and standard deviation for the medio-lateral pressure ratio during FFC, FMC, FFF, HO and LFC are listed in table 4.

**Table 4:** Mean, standard deviation and p-value for the t-test or Mann-whitney U-test (MWU-test) ( $\alpha = 0.1$ ) and logistic regression analysis ( $\alpha = 0.05$ ) of the medio-lateral pressure ratio at first foot contact (FFC), first metatarsal contact (FMC), forefoot flat (FFF), heel off (HO) and last foot contact (LFC).

	Mean control group	SD control group	Mean PFP group	SD PFP group	Significance t-test MWU-test	Significance Logistic regression
Ratio FFC	0.898	0.266	0.806	0.236	0.082 <sup>+</sup>	0.003*
Ratio FMC	1.155	0.224	1.171	0.231	0.386	/
Ratio FFF	1.114	0.324	1.177	0.305	0.130	/
Ratio HO	1.538	0.747	1.691	0.698	0.143	/
Ratio LFC	33.880	53.499	25.488	31.577	0.760	/

(PFP= patellofemoral pain group, \*p  $\leq$  0.05, <sup>+</sup> p  $\leq$  0.1).

Logistic regression analysis revealed that the maximal velocity of the change in latero-medial direction (X-component) of the COP during the forefoot contact phase was significantly slower ( $P = 0,002$ ) in the subjects who developed patellofemoral pain compared to the control group. The COP shifted less quick from the lateral to the medial side of the foot in the group who developed patellofemoral pain. The mean value of the X- component of the centre of pressure showed that during the FFCP the X-component of the COP was more laterally directed in PFP group (mean X-comp FFCP =  $-0,559 \pm 1,495$ ) and more medially directed in the control group (mean X-comp FFCP =  $0,107 \pm 1,356$ ) (A positive value indicates a pressure medially directed of the heel- M2 axis, a negative value indicates a pressure laterally directed of the heel- M2 axis). However, this difference of the X-component of the COP during the FFCP between the recruits who developed patellofemoral pain and the control group was not statistically significant ( $P = 0,088$ ) (table 5).

**Table 5:** Mean, standard deviation and p-value for the t-test ( $\alpha = 0.1$ ) and logistic regression analysis ( $\alpha = 0.05$ ) of the X-component (medio-lateral) of the COP during the Initial contact phase (ICP), Forefoot contact phase (FFCP), Foot flat phase (FFP), Forefoot push off phase (FFPOP).

	Mean control group	SD control group	Mean PFP group	SD PFP group	Significance t-test	Significance Logistic regression
X-comp ICP	- 0.300	1.101	- 0.352	2.359	0.672	/
X-comp FFCP	0.107	1.356	- 0.559	1.495	0.088 <sup>+</sup>	0.383
X-comp FFP	0.039	5.576	- 0.192	5.160	0.755	/
X-comp FFPOP	- 0.526	5.162	- 1.267	15.454	0.528	/

PFP= patellofemoral pain group, a positive value indicates a pressure medially directed of the heel- M2 axis, a negative value indicates a pressure laterally directed of the heel- M2 axis. (<sup>+</sup> p  $\leq$  0.1).

The mean and standard deviation for the maximal velocity of the X-component of the COP during the four phases (ICP, FFCP, FFP, FFPOP) are shown in table 6.

**Table 6:** Mean, standard deviation and p-value for the t-test or Mann-whitney U-test (MWU-test) ( $\alpha = 0.1$ ) and logistic regression analysis ( $\alpha = 0.05$ ) of the maximal velocity of the X-component of the COP during the Initial contact phase (ICP), Forefoot contact phase (FFCP), Foot flat phase (FFP), Forefoot push off phase (FFPOP).

	Mean control group	SD control group	Mean PFP group	SD PFP group	Significance t-test MWU-test	Significance Logistic regression
MAXvx ICP	135.124	49.808	141.842	81.268	0.875	/
MAXvx FFCP	79.500	39.789	49.210	29.744	0.001 <sup>+</sup>	0.002*
MAXvx FFP	106.736	52.039	101.329	43.076	0.814	/
MAXvx FFPOP	240.966	121.141	198.409	184.231	0.055 <sup>+</sup>	0.793

(PFP= patellofemoral pain group, MAXvx = maximal velocity of the X-component of the COP, \* $p \leq 0.05$ , <sup>+</sup> $p \leq 0.1$ ).

## DISCUSSION

Although the patellofemoral pain syndrome is one of the most prevalent musculoskeletal injuries encountered in sports medicine<sup>1, 2, 3, 4, 5, 6, 7</sup> there is no clarity concerning the aetiological risk factors which predispose people to develop this disorder. A number of theories including patellofemoral malalignment, soft periarticular tissue imbalances, quadriceps muscle weakness, vasti muscle imbalance and bony abnormalities<sup>7, 10, 13</sup> have been propounded regarding the aetiology of PFP.

In this study, logistic regression analyses of plantar pressure measurements during gait revealed three gait related intrinsic factors as being predicting factors for the development of patellofemoral pain. These factors are 1) a more laterally directed pressure distribution at initial foot contact 2) a shortened time to maximal pressure on the fourth metatarsal and 3) a delayed change of the COP in latero-medial direction during the forefoot contact phase of gait.

The results of the analysis of the medio-lateral pressure ratio showed that in the group who developed patellofemoral pain plantar pressure distribution was significantly more laterally directed at first foot contact compared to the control group. This could indicate that in the PFP group the foot pronates less at the initial contact phase of the gait pattern than in the non-injured group. In accordance to this finding Duffey et al.<sup>2</sup> also found that runners suffering from anterior knee pain had 25% less pronation during the first 10% of the support phase. As an adequate pronation of the foot is necessary to make an appropriate shock-absorption possible as the foot strikes the ground, less pronation may cause a more rigid landing and thereby increase the shock to the lower extremity contributing to overuse injury.

In addition to the medio-lateral pressure distribution, this study also identified the time to maximal pressure on the fourth metatarsal and the maximal velocity of the change of the COP in latero-medial direction during the FFCP as intrinsic risk factors for the development of patellofemoral pain.

In the group who developed anterior knee pain during this study, maximal pressure on the fourth metatarsal was reached earlier compared to the control group. Though, the maximal velocity of the change of the centre of pressure in latero-medial direction during the FFCP was significantly slower than in the control group. Thus, in the subjects who developed PFP maximal pressure on the fourth metatarsal occurred sooner but more time was needed during the forefoot contact phase to shift the COP from the lateral side to the medial side of the foot. This suggests that in this study the persons who developed anterior knee pain roll over their foot more on the lateral side compared to the control group. Additionally, however not statistically significant ( $P = 0,088$ ), during the forefoot contact phase the X-component of the COP was more laterally directed to the heel-M2 axis of the foot in the PFP group compared to a more medially directed X-component, relative to the heel-M2 axis, in the control group. Furthermore, however not statistically significant, the X-component of the COP in the PFP group also appeared to be directed more on the lateral side of the foot during the initial contact phase, foot flat phase and forefoot push off phase (table 5).

The fact that in the PFP group initial contact of the foot with the ground during gait occurred more on the lateral side of the foot and the centre of pressure shifted slower from the lateral to the medial side of the foot during foot rollover may cause less shock absorption in the foot. Consequently a greater part of the ground reaction forces are transferred to the more proximal joints including the knee. This could result into a higher load on the patellofemoral joint and consequently to overload of the patellofemoral joint which leads to patellofemoral pain.

In addition, the more laterally directed pressure suggests a less pronated position of the foot during the rollover pattern during gait which could lead to less internal rotation of the tibia. This could place the tibial tuberosity in a more lateral position relative to the femur and thereby increases the Q angle<sup>6</sup>. A larger Q angle would tend to create a larger lateral vector and potentially a greater predisposition to lateral patellar tracking when compared to a smaller Q angle, thereby increasing the contact pressure on the facets of the patella ipsilateral to the direction of the tibial rotation<sup>6, 29</sup>.

An intervention which should be addressed in future studies is the use of foot orthotics in persons showing aberrations of the rollover pattern of the foot identified in this study. In the literature it has been suggested that patients with patellofemoral pain may benefit from foot

orthoses if they demonstrate signs of excessive foot pronation<sup>31, 32, 33</sup>. This may also be true for persons with PFP showing an excessive laterally oriented rollover pattern of the foot. By correcting the aberrant motion of the foot, foot orthoses may be a valuable adjunct to other curative or preventative intervention strategies for patellofemoral pain. However, future studies are needed to determine the effectiveness of orthoses in the treatment of PFP by correcting the alignment profile which, in this study, has been identified to be predisposing for the development of patellofemoral pain.

In the current study 36 of the 84 recruits (43%) developed patellofemoral pain, which is remarkably high. Of these 36 recruits who developed PFP, three additionally developed shin splints (1 unilateral, 2 bilateral) and three other recruits additionally developed an overuse injury of the Achilles tendon (bilateral). In the group of recruits which did not develop PFP, two recruits developed shin splints (unilateral) and four developed an overuse injury of the Achilles tendon (2 unilateral, 2 bilateral).

One reason for the high incidence of PFP could be attributed to the intensity of the training exercises (marching with heavy loaded backpacks, roadwork, running) the recruits went through during the 6 week training period. The very intense exercises with little time to recover could explain the high amount of subjects who developed anterior knee pain by overuse. Therefore, care must be taken when comparing the results of this population with results from groups with a different exercise status.

In this study the group who developed patellofemoral complaints seemed to show a roll over pattern of the foot which occurred more on the lateral side than the group who did not develop PFP. Previous studies, however, suggested that there is an association between excessive rearfoot pronation and anterior knee pain<sup>13, 14, 16, 17, 30</sup>. The results of this study may contradict the results of former studies. However, in our opinion, it seems plausible that when the normal physiological loading of the joint is exceeded by overuse both deviations of the normal roll over pattern of the foot, excessive pronation as well as insufficient pronation, may lead to patellofemoral dysfunction and hence complaints. In this way both abnormalities of the gait pattern may cause patellofemoral complaints by different kinds of aberrations of the normal biomechanics of the lower limb when the knee joint is exposed to overuse.

An important difference between our study and previous studies is the fact that this study is the first to prospectively examine potential gait related risk factors for patellofemoral pain. Former research assessed potential aetiological factors for PFP in a retrospective way.

However, to investigate cause and effect-relationships it is necessary that potential risk factors for an injury are examined in subjects before the occurrence of injury.

Another difference of current study with previous studies, when comparing the methods used to analyze the gait pattern, is that former studies used two or three dimensional kinematic analysis and force platform measurements to determine rearfoot motion and ground reaction forces whereas in this study a footscan<sup>®</sup> pressure plate was used to measure plantar pressure and plantar pressure distribution underneath the sole of the foot. The plantar pressure measurements allow an indirect interpretation of the rollover pattern of the subject's foot. Previous research<sup>20</sup> has shown that plantar pressure measurements have a good predictive value for exercise-related lower leg injuries. Subjects were also asked to walk barefoot so that discrepancies due to unusual footwear were controlled.

In conclusion, based on plantar pressure measurements during barefoot walking, the results of our study revealed three potential gait related intrinsic risk factors for the development of patellofemoral pain i.e. a more laterally directed pressure distribution at initial foot contact, a shortened time to maximal pressure on the fourth metatarsal and a delayed change of the COP in latero-medial direction during the forefoot contact phase of gait. These findings suggest that during gait the feet of the persons who developed anterior knee pain in this study had a heel strike in a less pronated position and rollover more on the lateral side compared with the control group.

Accurate measurement of the motion of the foot should be considered as an important part of the assessment of persons with anterior knee pain in addition to the local assessment of the knee (e.g. patellofemoral alignment, tension of soft tissue structures, periarticular structures, strength of quadriceps muscle, vasti muscle coordination). However, in order to perform an accurate evaluation of the rollover pattern of the foot, in most cases a measuring device such as a pressure plate will be needed as often aberrations of foot rollover, as seen in this study, may not be that obvious they could be perceived from a clinical observation of gait. When the assessment of the rollover pattern of the foot is taken into account in the examination of PFP-patient, the results of this study can be considered as valuable in identifying persons at risk for patellofemoral pain.

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## REFERENCES

1. Brechter JH, Powers CM. Patellofemoral stress during walking in persons with and without patellofemoral pain. *Med Sci Sports Exerc.* 2002; 34 (10): 1582-1593.
2. Duffey MJ, Martin DF, Cannon DW, et al. Etiologic factors associated with anterior knee pain in distance runners. *Med Sci Sports Exerc.* 2000; 32 (11): 1825-1832.
3. Fulkerson JP. The etiology of patellofemoral pain in young active patients: A prospective study. *Clin Orthop.* 1983; 179: 129-133.
4. Levinger P, Gilleard W. An evaluation of the rearfoot posture in individuals with patellofemoral pain syndrome. *J Sports Sci and Med.* 2004; 3: 8-14.
5. Messier SP, Davis SE, Curl WW, et al. Etiologic factors associated with patellofemoral pain in runners. *Med Sci Sports Exerc.* 1991; 23 (9): 1008-1015.
6. Powers CM. The influence of altered lower-extremity kinematics on patellofemoral joint dysfunction: A theoretical Perspective. *J Orthop Sports Phys Ther.* 2003; 33: 639-646.
7. Powers CM, Chen PY, Reischl SF, et al. Comparison of foot pronation and lower extremity rotation in persons with and without patellofemoral pain. *Foot ankle Int.* 2002; 23 (7): 634-40.
8. Bennel K, Bartam S, Crossley K, et al. Outcome measures in patellofemoral pain syndrome: test retest reliability and inter-relationships. *Phys Ther in Sports.* 2000; 1: 32-41.
9. Johnston LB, Gross MT. Effects of foot orthoses on quality of life for individuals with patellofemoral pain syndrome. *J Orthop Sports Phys Ther.* 2004; 34(8): 440-447.
10. Witvrouw E, Lysens R, Bellemans J, et al. Intrinsic risk factors for the development of anterior knee pain in an athletic population: A two-year prospective study. *Am J Sports med.* 2000; 28 (4): 480-489.
11. Laprade J, Culham E. Radiographic measures in subjects who are asymptomatic and subjects with patellofemoral pain syndrome. *Clin Orthop and Related research.* 2003; 414: 172-182.
12. Cutbill JW, Ladly KO, Bray RC, et al. Anterior knee pain: A review. *Clin J Sport Med.* 1997; 7: 40-45.
13. Callaghan MJ, Baltzopoulos V. Gait analysis in patients with anterior knee pain. *Clin Biomech.* 1994; 9: 79-84.
14. Cheung RTH, Ng GYF, Chen BFC. Association of footwear with patellofemoral pain syndrome in runners. *Sports Med.* 2006; 36(3): 199-205.

15. Buchbinder MR, Napora NJ, Biggs EW. The relationship of abnormal pronation to chondromalacia of the patella in distance runners. *J Am Podiatry Assoc.* 1979; 69: 159-162.
16. Eng JJ, Pierrynowski MR. Evaluation of soft foot orthotics in the treatment of patellofemoral pain syndrome. *Phys Ther.* 1993; 73(2): 62-68.
17. Hamill J, Bates BT, Holt KG. Timing of lower extremity joint actions during treadmill running. *Med Sci Sports Exerc.* 1992; 24(7): 807-13.
18. Tiberio D. The effect of excessive subtalar joint pronation on patellofemoral mechanics: A theoretical model. *J Orthop Sports Phys Ther.* 1987; 9: 160-165.
19. Murphy DF, Connolly DAJ, Beynon BD. Risk factors for lower extremity injury: a review of the literature. *Br J Sports Med.* 2003; 37: 13-29.
20. Willems TM, De Clercq D, Delbaere K, et al. A prospective study of gait related risk factors for exercise-related lower leg pain. *Gait and Posture.* 2006; 23(1): 91-98.
21. Clarke TE, Frederick EC, Hamill C. The study of rearfoot movement. Frederick EC, ed. *Running in sport shoes and playing surfaces.* Champaign, Illinois: Human Kinetics Publishers; 1984: 166-189.
22. Willems TM, Witvrouw E, De Cock A, et al. Gait related risk factors for exercise-related lower leg pain during shod running. *Med Sci Sports Exerc.* 2007; 39 (2): 330-339.
23. De Cock A, De Clercq D, Willems T, et al. Temporal characteristics of foot roll-over during barefoot jogging: reference data for young adults. *Gait and Posture.* 2005; 21(4): 432-439.
24. De Cock A, Willems TM, Witvrouw E, et al. A functional foot type classification with cluster analysis based on plantar pressure distribution during jogging. *Gait and Posture.* 2006; 23 (3): 339-347.
25. De Haven KE, Dolan WA, Mayer PJ. Chondromalacia patellae in athletes: clinical presentation and conservative management. *Am J Sports Med.* 1979; 7: 5-11.
26. Kannus P, Niitymaki S. Which factors predict outcome in the non operative treatment of patellofemoral pain syndrome? A prospective follow-up study. *Med Sci Sports Exerc.* 1994; 26: 289-296.
27. Menz HB. Two feet or one person? Problems associated with statistical analysis of paired data in foot and ankle medicine. *The foot.* 2004; 14: 2-5.
28. Hosmer DW, Lemeshow S. *Applied logistic regression.* New York: Wiley; 1989: 1-353.

29. Lee TQ, Morris G, Csintalan RP. The influence of tibial and femoral rotation on patellofemoral contact area and pressure. *J Orthop Sports Phys Ther.* 2003; 33(8): 686-693.
30. Areblad M, Nigg PM, Ekstrand J, et al. 3 Dimensional measurement in rearfoot running. *J Biomech.* 1990; 23(9): 933-40.
31. GrossMT, Foxworth JL. The role of foot orthose as an intervention for patellofemoral pain. *J Orthop Sports Phys Ther.* 2003; 33(11): 661-670.
32. Eng JJ, Pierrynowski MR. Evaluation of soft foot orthotics in the treatement of patellofemoral pain syndrome. *Phys Ther.* 1993 73(5): 62-68.
33. Klingman RE, Liaos SM, Hardin KM. The effect of subtalar joint posting on patellar glide position in subjects with excessive rearfoot pronation. *J Orthop Sports Phys Ther.* 1997; 25(3): 185-191.



## **CHAPTER 3**

### **Gait related intrinsic risk factors for patellofemoral pain in novice recreational runners**

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## **Abstract**

**Objective:** The purpose of this study was to prospectively determine gait related intrinsic risk factors for patellofemoral pain (PFP) in a population of novice recreational runners.

**Design:** Prospective cohort study.

**Participants:** One hundred and two novice recreational runners (89 women, 13 men), with no history of knee or lower leg complaints.

**Interventions:** The subjects' standing foot posture was examined and plantar pressure measurements during running were collected. Subsequently, the subjects participated in a ten week 'start to run' program. During this period all sports injuries were registered by a sports medicine physician.

**Main Outcome Measurements:** The relationship between the standing foot posture and PFP was investigated and gait related intrinsic risk factors for PFP were determined.

**Results:** The 17 runners who developed PFP in this study exerted a significantly higher vertical peak force underneath the lateral heel and metatarsals 2 and 3. Logistic regression analysis showed that a significantly higher vertical peak force underneath the second metatarsal and shorter time to the vertical peak force underneath the lateral heel were predictors for PFP. No significant evidence was found for an association between an excessively pronated or supinated foot posture and the development of PFP.

**Conclusions:** The findings suggest that an excessive impact shock during heel strike and at the propulsion phase of running may contribute to an increased risk of developing PFP. The hypothesis that persons at risk for PFP show an altered static foot posture in comparison with non-afflicted persons is not supported by the results of this study.

**Keywords:** patellofemoral pain; kinetic analysis; foot posture; running injuries

## INTRODUCTION

The growing interest in disease prevention has lead to an increased participation of the public in sports during the recent decades. [1, 2] Due to its easy accessibility, distance running is practised by many people and increases in popularity. [2] The increased participation in sports leads to an increase in sports related injuries. Runners continue to be among the most commonly injured athletes. [32] Studies estimate the yearly incidence of running injuries to be 37-56%. [5, 19]

Epidemiological studies have found that in runners most of the injuries are located in the knee, with patellofemoral pain syndrome being the most prevalent injury reported in running. [2, 3, 4, 5, 20, 21] Due to the growing interest of the public in running, novice recreational runners are becoming a considerable population at risk for the development of anterior knee pain.

Despite the high incidence of patellofemoral pain (PFP) in competitive and recreational athletes [6, 7, 8] there is still a lack of consensus concerning the aetiological mechanisms of this disorder. Focussing on the structures around the knee joint, factors such as malalignment of the patellofemoral joint, soft periarticular tissue imbalances, weakness of the quadriceps muscle, vasti muscle imbalance and bony abnormalities have been identified as potential risk factors for the development of PFP.[8, 9, 10]

However, it has been recognised that the mechanics of the patellofemoral joint is also influenced by segmental interactions of the lower extremity.[12] Consequently, abnormal kinematics of the ankle and foot have been propounded to be one of the possible risk factors for PFP.[9]

A number of studies have investigated foot motion as a risk factor for patellofemoral pain with a focus on abnormal pronation of the foot and subsequent lower extremity rotation. [9, 10, 11, 12, 13, 14, 15, 16, 17, 18] However, based upon a review of the literature, there is no consensus whether an abnormal pronation of the foot is an aetiological factor for anterior knee pain. One of the most important reasons for this lack of consensus is probably a lack of prospective studies designed to determine risk factors for PFP. Most studies which have been investigating the relationship between foot movement and PFP are retrospective or theoretically based. [9, 10, 11, 12, 13, 14, 15, 16, 17, 18] Because of their retrospective

design, it is unclear if the deficits found in these studies are a cause or a consequence of injury.

With the current emphasis on injury prevention, studies designed to examine potential intrinsic risk factors for patellofemoral pain before the occurrence of injury are imperative.

Therefore, the purpose of this investigation was to perform a comprehensive, prospective study on gait related intrinsic risk factors for PFP in a population of novice recreational runners. It was hypothesised that subjects who develop PFP show an altered rollover pattern and posture of the foot in clinical measurements.

## **MATERIALS AND METHODS**

### **Subjects**

One hundred and forty-three novice recreational runners, who were enrolled in a ten week 'start to run' program organised by a Belgian track and field club in April 2007, agreed to participate in the study. Of these 143 potential participants, 14 had a history of a surgical procedure involving the knee, lower leg, ankle or foot or a history of an injury to the knee, lower leg, ankle or foot that caused them to seek medical attention within six months before the start of the study and were excluded from the study. Consequently, 129 runners (107 women, 22 men) were prospectively examined. Twenty-seven out of the originally 129 tested subjects quit the 'start to run' program due to reasons other than the development of an injury (social or work related reasons) and consequently dropped out of the study.

The remaining 102 runners had an average age of 37 years ( $SD \pm 9.50$ ), an average height of 165 cm ( $SD \pm 25$ ), an average weight of 69 kg ( $SD \pm 15$ ) and an average Body Mass Index (BMI) of 25 ( $SD \pm 3$ ). The aim of the study was explained to each subject and they all signed an informed consent. The study was approved by the ethical committee of the Ghent University Hospital.

### **Evaluation**

Before the initiation of the 'start to run' program the subjects' static standing foot posture was evaluated using the Foot Posture Index<sup>®</sup>. The roll-over pattern of the subjects' feet was examined during running using a footscan pressure plate (RsScan International).

## **Evaluation of standing foot posture**

The standing position of the subjects' feet was evaluated using the Foot Posture Index (FPI). The foot posture index is a validated clinical tool for quantifying the degree to which a foot can be considered to be in a pronated, supinated or neutral position. [25] It is intended to be a simple method of scoring various features of foot posture into a quantifiable result, which gives an indication of the overall foot posture.

According to the guidelines of Redmond et al. [25] the subjects were asked to stand on a platform in a relaxed stance position with double limb support, their arms by the side and looking straight ahead. By palpation and a series of observations, the weightbearing foot posture was rated according to a series of predefined criteria. The six clinical criteria employed in the FPI were: 1) palpation of the talar head, 2) observation of the supra and infra lateral malleolar curvature, 3) observation of the calcaneal frontal plane position, 4) observation of prominence in the region of the talonavicular joint, 5) observation of the congruence of the medial longitudinal arch and 6) observation of abduction/adduction of the forefoot on the rearfoot. Each of the component tests were graded 0 for neutral, -1 for moderate signs of supination, -2 for clear signs of supination, +1 for moderate signs of pronation and +2 for clear signs of pronation. When the scores of each test were combined, the aggregate value gave an estimate of the overall foot posture. High positive aggregate values indicated a pronated posture (pronated = +6 to +9, highly pronated = 10+), high negative aggregate values indicated a supinated overall foot posture (supinated = -1 to -4, highly supinated = -5 to -12) and an aggregate score from 0 to +5 indicated a neutral foot. Each foot was scored independently. All of the subjects were evaluated by the same examiner.

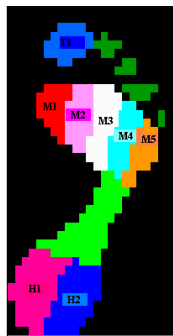
## **Plantar pressure measurements**

Before the start of the training period all subjects underwent plantar pressure measurements during barefoot running. By running barefoot, discrepancies due to unusual footwear were controlled. Clarke et al.[22] stated that in running analysis barefoot is an accepted baseline state and it has been shown that gait related risk factors for exercise related lower leg pain are more distinct in the barefoot condition compared to the shod condition [23]. A footscan pressure plate (RsScan International, 2m x 0,4m, 480 Hz) was mounted in the middle of a 15 m long walkway. The walkway was covered by a thin rubber mat so that the pressure plate would not be visible to the subjects.

The subjects were asked to run at a self chosen comfortable moderate velocity along the walkway. All subjects were allowed to familiarize themselves with the procedures before data collection. Three valid left and right stance phases were measured. A trial was considered to be valid when there was a heel strike pattern, no adjustment in step length or frequency to aim on the pressure plate. De Cock et al. [24] found that temporal plantar pressure variables, measured with the footscan<sup>®</sup> pressure plate are reliable ( $ICC > 0.75$ ).

## Data analysis

For each trial of the plantar pressure measurements, the footprint was divided into eight anatomical areas (figure 1). These eight zones were defined as the medial heel (H1), lateral heel (H2), metatarsals I – V (M1, M, M3, M4, M5) and the hallux (T1).



**Figure 1:** Location of the eight anatomical areas. (RsScan International)

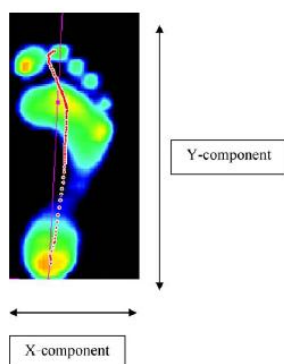
Temporal data (time to peak force, instants on which the regions make contact and instants on which the regions end contact), peak force data and absolute impulses (mean force x loaded contact time) and relative impulses (absolute impulse x 100 / sum of all impulses) were calculated for all eight regions. The total foot contact time and five distinct instants of foot roll-over were determined for each trial. These five instants of foot roll-over were: first foot contact (FFC), first metatarsal contact (FMC), forefoot flat (FFF), heel off (HO) and last foot contact (LFC). FFC was defined as the instant the foot made first contact with the pressure plate. FMC was defined as the moment when one of the metatarsal heads contacted the pressure plate. FFF was defined as the first instant all metatarsal heads made contact with the plate. HO was defined as the instant the heel region ended contact with the plate. LFC was defined as the last contact of the foot on the plate. Based on these instants, total foot contact could be divided into four phases: initial contact phase (ICP = FFC to FMC), forefoot contact

phase (FFCP = FMC to FFF), foot flat phase (FFP = FFF to HO) and forefoot push off phase (FFPOP = HO to LFC).

A medio-lateral force ratio  $[(M1 + M2 + H1)/(M4 + M5 + H2)]$  was calculated at the five instants of foot contact. This ratio describes the force distribution in the foot at the five instants of foot contact. Excursion ranges of this ratio were calculated over the four phases (ICP, FFCP, FFP, FFPOP).

The X-component (medio-lateral) and Y-component (anterior-posterior) of the centre of force (COF) scaled to the foot width and foot length, respectively, were analyzed (figure 2). The positioning and displacements of the components were calculated at the five instants and in the four phases.

The mean of all kinetic data was taken from the three trials. De Cock et al. have shown that the mean of three trials is sufficient for analysis. [24, 26]



**Figure 2:** The X-component (medio-lateral) and Y-component (anterior-posterior) of the COF. (RsScan International)

### **‘Start to run’ program**

The subjects, included in this study, were all untrained novice runners at the start of a ten week during ‘start to run’ program. During this ‘start to run’ program the subjects trained three times a week following a fixed schedule with the aim of being able to run a distance of five kilometres at the end of the 10th week of the training program. The training sessions were all group sessions under supervision of an experienced track and field trainer. The subjects who participated in this study did not practise any other kind of sports-activities during the course of the start to run program.

## **Registration of injuries**

During the ten week 'start to run' program, patellofemoral complaints were diagnosed and registered by the same sports medicine physician. The subjects were able to freely consult this sports medicine physician, who was present at each training-session. To be considered as a patellofemoral pain patient, subjects had to have a characteristic history and symptoms of patellofemoral pain syndrome and exhibit two of the following clinical criteria on assessment: pain on direct compression of the patella against the femoral condyles with the knee in full extension, tenderness of the posterior surface of the lateral or medial rim of the patella on palpation, pain on resisted knee extension, pain with isometric quadriceps muscle contraction against suprapatellar resistance with the knee in 15° of flexion. In addition, subjects had to have negative findings in the examination of knee ligaments, menisci, bursae, synovial plicae, Hoffa's fat pad, iliotibial band, the hamstrings, quadriceps and patellar tendons and their insertions. Previous studies indicate that these criteria are sensitive and specific for diagnosing patellofemoral pain. [8, 27, 28]

## **Statistical analysis**

Statistical analysis was performed with SPSS for windows version 12.0 (SPSS Inc, Chicago, Ill). A binary logistic regression analysis [29] was performed to identify gait related intrinsic risk factors for patellofemoral pain. Firstly, in order to reduce the number of variables, student's *t*-tests (if the distribution of the data was normal) or Mann-Whitney U-tests (if no normal data distribution was obtained) were undertaken for examining possible differences between the group which developed patellofemoral pain and the non-symptomatic group, for each of the test variables. All variables showing a P-value < 0,1 in the univariate analysis were entered separately into the logistic regression analysis.

The Pearson chi-square test was conducted to examine the relationship between the static standing foot posture and the development of patellofemoral problems. Statistical significance was accepted at the level of  $\alpha \leq 0.05$ .

## **RESULTS**

Seventeen (16 female and 1 male) of the 102 runners developed patellofemoral pain during the ten week 'start to run' program.



The anthropometrical characteristics of the 17 subjects who sustained patellofemoral problems were compared with those of the group without PFP. There were no significant differences between these two groups with respect to their average age, height, weight and BMI. The anthropometrical data of the PFP-group and the non-symptomatic group are presented in table 1.

**Table 1:** Mean, standard deviation (SD) and p-value for the t-test/ Mann-Whitney U-test (MWU-test) of the age, height, weight and Body Mass Index (BMI) of the group of runners with PFP and the group without PFP (asymptomatic group).

	Mean $\pm$ SD PFP group	Mean $\pm$ SD asymptomatic group	Significance t-test MWU-test
Age (years)	39.4 $\pm$ 10.3	37.6 $\pm$ 9.4	0.493
Height (cm)	164.5 $\pm$ 26.8	167.4 $\pm$ 7.5	0.572
Weight (kg)	69.3 $\pm$ 8.1	69.3 $\pm$ 15.8	0.985
BMI	24.9 $\pm$ 3.5	25.1 $\pm$ 2.8	0.803

( $\alpha = 0.05$ )

T-tests showed that the vertical peak force underneath metatarsals 2 ( $P = 0.016$ ) and 3 ( $P = 0.026$ ) and underneath the lateral heel ( $P = 0.034$ ) was significantly higher in the PFP-group compared to the non-symptomatic group.

Logistic regression analysis revealed a significantly ( $P = 0.037$ ) higher vertical peak force underneath the second metatarsal as a predisposing factor for patellofemoral pain (table 2).

**Table 2:** Mean, standard deviation, p-value for the t-test or Mann-Whitney U-test (MWU-test) ( $\alpha = 0.1$ ) and logistic regression analysis ( $\alpha = 0.05$ ) of the vertical peak force underneath the eight anatomical areas.

	Mean $\pm$ SD PFP group	Mean $\pm$ SD asymptomatic group	Significance t-test MWU-test	Significance Logistic regression	95% CI
Fmax T1 (N)	174.54 $\pm$ 68.58	151.67 $\pm$ 92.30	0.336	/	/
Fmax M1 (N)	244.85 $\pm$ 172.64	210.97 $\pm$ 118.52	0.324	/	/
Fmax M2 (N)	366.44 $\pm$ 121.94	286.97 $\pm$ 122.25	0.016 <sup>+</sup>	0.037*	-143.88-(-15.06)
Fmax M3 (N)	308.06 $\pm$ 69.43	245.77 $\pm$ 109.12	0.026 <sup>+</sup>	0.523	-116.99-(-7.57)
Fmax M4 (N)	239.79 $\pm$ 66.40	195.25 $\pm$ 90.89	0.058	/	/
Fmax M5 (N)	148.35 $\pm$ 78.65	115.30 $\pm$ 71.24	0.089	/	/
Fmax H1 (N)	478.34 $\pm$ 182.24	409.38 $\pm$ 211.21	0.213	/	/
Fmax H2 (N)	514.68 $\pm$ 255.78	402.46 $\pm$ 218.63	0.034 <sup>+</sup>	0.872	-230.81-6.38

(PFP= patellofemoral pain, Fmax= max force, N= Newton, \* $p \leq 0.05$ , <sup>+</sup>  $p \leq 0.1$ ), 95% CI = 95% confidence interval.

For the temporal data, t-tests indicated that the time to the vertical peak force underneath H2 and H1 ( $P = 0.037$  and  $P = 0.016$ , respectively), relative to the total time of foot contact, was significantly shorter in the PFP-group compared to the non-symptomatic group.

Logistic regression showed a significantly shorter ( $P = 0.048$ ) time to the vertical peak force underneath the lateral heel as a predisposing factor for patellofemoral pain (table 3). Logistic regression did not identify significant differences for any other temporal parameters.

**Table 3:** Mean, standard deviation,  $p$ -value for the  $t$ -test or Mann-Whitney  $U$ -test (MWU-test) ( $\alpha = 0.1$ ) and logistic regression analysis ( $\alpha = 0.05$ ) of the time (sec) to the vertical peak force underneath T1, M1-M5, H1 and H2, relative to the total time of foot contact.

	Mean $\pm$ SD PFP group	Mean $\pm$ SD asymptomatic group	Significance $t$ -test MWU-test	Significance Logistic regression	95% CI
ReltFmax T1	0.717 $\pm$ 0.071	0.713 $\pm$ 0.060	0.798	/	/
ReltFmax M1	0.562 $\pm$ 0.049	0.578 $\pm$ 0.052	0.251	/	/
ReltFmax M2	0.578 $\pm$ 0.040	0.574 $\pm$ 0.040	0.693	/	/
ReltFmax M3	0.548 $\pm$ 0.040	0.551 $\pm$ 0.039	0.776	/	/
ReltFmax M4	0.515 $\pm$ 0.034	0.505 $\pm$ 0.041	0.356	/	/
ReltFmax M5	0.480 $\pm$ 0.041	0.461 $\pm$ 0.057	0.201	/	/
ReltFmax H1	0.061 $\pm$ 0.042	0.081 $\pm$ 0.043	0.016 <sup>+</sup>	0.350	-.002-.04
ReltFmax H2	0.040 $\pm$ 0.018	0.054 $\pm$ 0.026	0.037 <sup>+</sup>	0.048*	.001-.03

(PFP= patellofemoral pain group, \* $p \leq 0.05$ , <sup>+</sup> $p \leq 0.1$ ), 95% CI = 95% confidence interval

There were no significant differences between the runners who developed PFP and the non-symptomatic runners for the medio-lateral force distribution at the five instants and four phases of foot contact during foot roll-over.

The Pearson's chi-square test showed no relationship ( $P = 0.788$ ) between the presence of a neutral, pronated, highly pronated, supinated or highly supinated static foot posture and the development of patellofemoral problems in the investigated population. The distribution of the subjects of the PFP-group and non-symptomatic group into the five categories of the FPI is presented in table 4.

**Table 4:** Distribution (exact amount and in %) of the subjects of the patellofemoral pain (PFP) group and the asymptomatic group in the five categories (normal, pronated, highly pronated, supinated or highly supinated) of the foot posture index (FPI).

FPI	PFP group (n)	PFP group (%)	Asymptomatic group (n)	Asymptomatic group (%)
Normal	9	52.9	50	58.3
Pronated	6	35.3	22	26.2
Highly pronated	0	0	3	3.6
Supinated	2	11.8	7	8.3
Highly supinated	0	0	3	3.6
Total	17	100	85	100

(n = exact amount)

No differences with the current results concerning the analysis of the running foot roll-over patterns and the relationship between the standing foot posture and the development of PFP

were found when the group of women, which formed the majority of the investigated population, was considered solely.

## **DISCUSSION**

Motions of pronation and supination occur during the roll-over pattern of the foot and assist in the normal locomotion during walking and running. Aberrations of these motions however have been suggested to contribute to the pathomechanics of patellofemoral pain. [1, 10, 12, 16, 17, 30] An excessive pronation of the subtalar joint is an abnormality which has been debated as being a possible risk factor for the development of PFP. However, a consensus has not been reached concerning this issue in the current literature. [1, 9, 10, 12, 16, 17, 30, 31]

The purpose of this investigation was to determine, in a prospective way, if a certain roll-over pattern and static posture of the foot predisposes recreational runners to the development of anterior knee pain.

In the population of recreational runners who participated in this study anterior knee pain was the most common sustained injury (17%), followed by shinsplints (11%) and Achilles tendon overuse injury (10%). In order of frequency, the other sustained injuries were overuse of the ankle joint (6%), Iliotibial band friction syndrome (4%), adductor injuries (3%), ankle inversion injuries (3%), patellar tendinitis (1%) and meniscal injury (1%). The incidence of patellofemoral problems in this population of novice runners is in accordance with previous studies which have reported the knee as the most common site of overuse injury in runners, with patellofemoral problems predominating. [2, 3, 4, 5, 12, 19, 20, 21, 33]

The results of this study showed that the runners who developed patellofemoral problems exerted a significantly higher vertical peak force underneath the lateral heel and underneath the second and third metatarsal during running. The results indicate that an excessive vertical peak force at the lateral side of the heel during heel impact and an excessive vertical peak force underneath M2 and M3 during the propulsion phase of running may be discriminating factors between runners afflicted with PFP and asymptomatic runners. Our results are similar to the findings of Callaghan and Baltzopoulos [10] who also found that runners suffering from patellofemoral pain exerted a significantly higher maximum vertical force at heel impact and a significantly higher maximum vertical propulsive force than non-injured runners. In this current study, logistic regression analysis identified a significantly higher vertical peak force underneath the second metatarsal as being a predicting factor for the development of

patellofemoral problems in the investigated population. During the roll-over pattern of the foot the peak pressures for the metatarsal areas occur during the forefoot push off phase of foot roll-over. [24] According to a study by De Cock et al. [24], the rise to peak pressure starts laterally at the fifth metatarsal, followed by a synchronous push off pattern of M4, M3 and M1. Finally, the second metatarsal reaches its maximal pressure at approximately 62 % of total foot contact and is the last metatarsal reaching its peak pressure and leaving the ground. [24] Our finding is in accordance with the findings of Messier et al. [14] who also demonstrated a significantly higher maximum vertical propulsive force in runners with PFP. Although in this current study the difference in vertical peak force between the PFP-group and asymptomatic group was only significant underneath the lateral heel and metatarsals 2 and 3, it is however remarkable that in the group of runners who developed PFP the vertical peak force values were higher underneath all eight anatomical areas (table 2).

In the runners who sustained PFP, the significantly higher vertical force at the lateral heel during the heel strike and at the second metatarsal near the end of the propulsion phase could cause that higher vertical forces are transferred to the more proximal joints such as the knee. This higher impact shock could cause an excessive load on the patellofemoral joint which eventually may lead to an overload of this joint, resulting in the development of patellofemoral pain.

In the runners who sustained PFP in this study, the vertical peak force at the medial and lateral heel at heel strike was reached sooner than in the non-symptomatic runners. Logistic regression identified a significantly shortened time to the vertical peak force at the lateral heel as the second gait related risk factor for the development of PFP in the investigated population. These results are in contrast with the findings of Messier et al. (1991) who did not find a significant difference in the time to the maximum vertical force at heel impact between runners with patellofemoral pain and uninjured runners. However in this current study logistic regression identified a relative shorter time to the vertical peak force at the lateral heel as an intrinsic risk factor for PFP, the odds ratio (which depicts the ratio of the odds of an event occurring in one group to the odds of it occurring in the other group) of this parameter was  $3.45 \times 10^{-14}$ . Because the odds ratio of this parameter approached zero, the odds that PF-problems in the PF-group arose as a result of a shortened time to the vertical peak force at the lateral heel, is very small. We therefore believe it would be wise to consider the significance of this parameter as a trend that requires further verification.

In previous retrospective or theoretically based studies some researchers have speculated on the relationship between excessive foot pronation and PFP. [1, 7, 15, 16, 18, 35]

In the present study, the mediolateral force distribution underneath the sole of the foot during foot roll-over was not seen as an intrinsic risk factor for patellofemoral pain. Hence, in the population of this study the results did not show signs of the presence of an abnormal dynamic foot pronation in the persons who sustained patellofemoral problems. It has been suggested that excessive pronation can lead to excessive tibial and femoral internal rotation and patellar displacement resulting in pain. However, Powers et al. [9] did not find significant differences with respect to the magnitude and timing of peak foot pronation between individuals with PFP and non-painful individuals. Also in a prospective study of Hetsroni et al. [31] no consistent association was found between static or dynamic parameters of foot pronation and the risk of anterior knee pain.

In the investigated population there was no significant evidence of an association between persons who showed a neutral, pronated, highly pronated, supinated or highly supinated static foot posture and the development of patellofemoral problems. In the literature there is controversy regarding the static posture of the foot and its contribution to patellofemoral problems. [8, 13, 14, 34] Powers et al. [34] reported that subjects with patellofemoral pain had a greater degree of rearfoot varus than assessed subjects without PFP. Similarly, Levinger and Gilleard [13] found that individuals with PFP had a significantly more inverted position in subtalar joint neutral measurements and more rearfoot valgus in relaxed standing posture compared to asymptomatic controls. In contrast, a prospective study of Witvrouw et al. [8] found no significant differences in foot types between persons with and without PFP. Also Messier et al. [14] found normal arched feet in subjects with patellofemoral pain. However Powers et al. [34] and Levinger and Gilleard [13] demonstrated that subjects with patellofemoral pain showed a more inverted position in subtalar joint neutral measurements, caution must be made in generalising this finding to the entire population of PFP-patients. Both authors indicated that the differences between the group means (approximate 1° and 2°, respectively) were only slight and can be debated despite the statistical significance. Also, other factors that can contribute to PFP were not controlled for in their studies. The results of this current study are in agreement with the former study of Witvrouw et al. [8] which does suspect that static foot posture may not be a predicting factor for the development of patellofemoral pain. The more inverted position of the subtalar joint in the PFP-group, reported by Powers et al. [34] and Levinger and Gilleard [13], was measured in subtalar joint neutral position during a non-weight bearing position while in this current study the foot posture was evaluated in a weight bearing position. The differences in methods may also partly account for the differences found between these studies.

In addition, Powers et al. and Levinger and Gilleard observed their findings in retrospective studies. A frequent question in retrospective studies is whether the findings are the result or the cause of the injury. The findings of the above-mentioned authors are however not supported by the results of the former prospective study of Witvrouw et al. [8] and this current prospective study.

## **CONCLUSION**

A significantly higher vertical propulsive peak force underneath M2 and a shortened time to the vertical peak force at the lateral heel were identified as predisposing factors for PFP. The runners who developed PFP also showed a significantly higher vertical peak force at the lateral heel and M3. However further verification is needed to determine the association between the latter described predisposing factor and the development of PFP, the results indicate that a significantly higher impact shock during running may contribute to the development of this disorder.

No significant evidence was found of an association between an excessively pronated or supinated foot posture or medio-lateral force distribution during foot roll-over and the development of PFP in the investigated population.

More future prospective studies are required to determine whether the examination of the foot posture and roll over pattern of the foot is an important addition to other clinical measurements to explore the underlying aetiology of patellofemoral pain.

## REFERENCES

1. Cheung RTH, Ng GYF, Chen BFC. Association of footwear with patellofemoral pain syndrome in runners. *Sports Med.* 2006; 36 (3): 199-205.
2. van Gent RN, Siem D, van Middelkoop M, *et al.* Incidence and determinants of lower extremity running injuries in long distance runners: a systematic review. *Br J Sports Med.* 2007; 41: 469-480.
3. Clement DB, Taunton JE, Smart GW, *et al.* A survey of overuse running injuries. *Hys Sportsmed* 1981; 9: 47-58.
4. Newel SG, Bramwell ST. Overuse injurie to the knee in runners. *Phys Sportsmed* 1984; 12: 81-92.
5. Van Mechelen W. Running injuries. A review of the epidemiological literature. *Sports Med* 1992; 14:320-35.
6. Bennel K, Bartam S, Crossley K, *et al.* Outcome measures in patellofemoral pain syndrome: test retest reliability and inter-relationships. *Phys Ther in Sports.* 2000; 1: 32-41.
7. Johnston LB, Gross MT. Effects of foot orthoses on quality of life for individuals with patellofemoral pain syndrome. *J Orthop Sports Phys Ther.* 2004; 34(8): 440-447.
8. Witvrouw E, Lysens R, Bellemans J, *et al.* Intrinsic risk factors for the development of anterior knee pain in an athletic population: A two-year prospective study. *Am J Sports med.* 2000; 28 (4): 480-489.
9. Powers CM, Chen PY, Reischl SF, *et al.* Comparison of foot pronation and lower extremity rotation in persons with and without patellofemoral pain. *Foot ankle Int.* 2002; 23 (7): 634-40.
10. Callaghan MJ, Baltzopoulos V. Gait analysis in patients with anterior knee pain. *Clin Biomech.* 1994; 9: 79-84.
11. Powers CM. The influence of altered lower-extremity kinematics on patellofemoral joint dysfunction: A theoretical Perspective. *J Orthop Sports Phys Ther.* 2003; 33: 639-646.
12. Duffey MJ, Martin DF, Cannon DW, *et al.* Etiologic factors associated with anterior knee pain in distance runners. *Med Sci Sports Exerc.* 2000; 32 (11): 1825-1832.
13. Lvinger P, Gilleard W. An evaluation of the rearfoot posture in individuals with patellofemoral pain syndrome. *J Sports Sci and Med.* 2004; 3: 8-14.
14. Messier SP, Davis SE, Curl WW, *et al.* Etiologic factors associated with patellofemoral pain in runners. *Med Sci Sports Exerc.* 1991; 23 (9): 1008-1015.

15. Buchbinder MR, Napora NJ, Biggs EW. The relationship of abnormal pronation to chondromalacia of the patella in distance runners. *J Am Podiatry Assoc.* 1979; 69: 159-162.
16. Eng JJ, Pierrynowski MR. Evaluation of soft foot orthotics in the treatment of patellofemoral pain syndrome. *Phys Ther.* 1993; 73(2): 62-68.
17. Hamill J, Bates BT, Holt KG. Timing of lower extremity joint actions during treadmill running. *Med Sci Sports Exerc.* 1992; 24(7): 807-13.
18. Tiberio D. The effect of excessive subtalar joint pronation on patellofemoral mechanics: A theoretical model. *J Orthop Sports Phys Ther.* 1987; 9: 160-165.
19. Wen DY, Puffer JC, Schmalzried TP. Lower extremity alignment and risk of overuse injuries in runners. *Med Sci Sports Exerc.* 1997; 29(10): 1291-1298.
20. Taunton JE, Ryan MB, Clement DB, *et al.* A retrospective case-control analysis of 2002 running injuries. *Br J Sports Med.* 2002; 36: 95-101.
21. Stefanyshyn DJ, Stergiou P, Lun VMY. Knee angular impulse as a predictor of patellofemoral pain in runners. *Am J Sports Med.* 2006; 34(11): 1844-1851.
22. Clarke TE, Frederick EC, Hamill C. The study of rearfoot movement. Frederick EC, ed. *Running in sport shoes and playing surfaces.* Champaign, Illinois: Human Kinetics Publishers; 1984: 166-189.
23. Willems TM, Witvrouw E, De Cock A, *et al.* Gait related risk factors for exercise-related lower leg pain during shod running. *Med Sci Sports Exerc.* 2007; 39 (2): 330-339.
24. De Cock A, De Clercq D, Willems T, *et al.* Temporal characteristics of foot roll-over during barefoot jogging: reference data for young adults. *Gait and Posture.* 2005; 21(4): 432-439.
25. Redmond AC, Crosbie J, Ouvrier RA. Development and validation of a novel rating system for scoring standing foot posture: The Foot Posture Index. *Clin Biomech.* 2006; 21: 89-98.
26. De Cock A, Willems TM, Witvrouw E, *et al.* A functional foot type classification with cluster analysis based on plantar pressure distribution during jogging. *Gait and Posture.* 2006; 23 (3): 339-347.
27. De Haven KE, Dolan WA, Mayer PJ. Chondromalacia patellae in athletes: clinical presentation and conservative management. *Am J Sports Med.* 1979; 7: 5-11.
28. Kannus P, Niitymäki S. Which factors predict outcome in the non operative treatment of patellofemoral pain syndrome? A prospective follow-up study. *Med Sci Sports Exerc.* 1994; 26: 289-296.



29. Hosmer DW, Lemeshow S. Applied logistic regression. New York: Wiley; 1989: 1-353.
30. Areblad M, Nigg PM, Ekstrand J, *et al.* 3 Dimensional measurement in rearfoot running. *J Biomech.* 1990; 23(9): 933-40.
31. Hetsroni I, Finestone A, Milgrom C, *et al.* A prospective biomechanical study of the association between foot pronation and the incidence of anterior knee pain among military recruits. *J Bone Joint Surg.* 2006; 88(7): 905-908.
32. Maclean Ch, McClay Davis I, Hamill J. Influence of a custom foot orthotic intervention on lower extremity dynamics in healthy runners. *Clin Biomech.* 2006; 21: 623-630.
33. Taunton JE, Ryan MB, Clement DB, *et al.* A prospective study of running injuries : the Vancouver Sun Run 'In Training' clinics. *Br J Sports Med.* 2003; 37: 239-244.
34. Powers CM, Maffucci R, Hampton S. Rearfoot posture in subjects with patellofemoral pain. *J Orthop Sports Phys Ther.* 1995; 22 (4): 155-160.
35. Klingman RE, Liaos SM, Hardin KM. The effect of subtalar joint posting on patellar glide position in subjects with excessive rearfoot pronation. *J Orthop Sports Phys Ther.* 1997; 25(3): 185-191.



## CHAPTER 4

### Relationship between hip strength and frontal plane posture of the knee during a forward lunge

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## Abstract

**Objective:** Excessive frontal plane knee movement during forward lunge movements might be associated with the occurrence of knee injuries in tennis. To determine whether hip muscle strength is related to the frontal plane motion of the knee during a functional lunge movement.

**Design:** A correlational study

**Participants:** 84 healthy subjects (76 men, 8 women), with no history of knee or lower leg complaints.

**Interventions:** Muscle strength of six hip muscle groups was measured using a hand-held dynamometer. Subjects were videotaped during a forward lunge and peak knee valgus or varus angles were determined using a digital video analysis software program.

**Main Outcome Measurements:** A correlation was examined between hip muscle strength and the amount of frontal plane movement of the knee during a forward lunge.

**Results:** There were no significant differences in hip muscle strength between the valgus group and the varus group during the forward lunge movement. No significant correlation was found between the strength of the assessed hip muscles and the amount of movement into valgus/varus. In the varus group a moderate positive correlation was found between the External Rotation/Internal Rotation force ratio and the amount of knee varus during the forward lunge movement ( $r = 0.31$ ,  $P = 0.03$ ).

**Conclusion:** The findings suggest that in healthy subjects hip muscle strength is not correlated to the amount of valgus/varus movement of the knee during a forward lunge. This suggests that other factors (e.g. proprioception, core hip stability) might be more important in controlling knee movement during this tennis-specific movement.

**Key words:** hip strength; frontal plane knee posture; forward lunge; knee injury; tennis

## INTRODUCTION

Tennis is one of the most popular sports world-wide. Due to the rising number of participants practicing tennis, the increasing pressure to practice, higher expectations of performance and, hence, increased demands on the human body, the injuries associated with tennis are becoming a matter of increasing concern in the world of sports medicine.[1] Modern tennis involves powerful movements which place a heavy load on the musculoskeletal system and hereby exposes tennis players to a high risk of overuse injuries [2]. Lower extremity injury occurs consistently more frequently than other injuries in tennis and other racquet sports.[3, 4, 5] Hereby, the knee accounts for the majority of lower limb injuries with tendon injuries, patellofemoral problems and intra-articular knee injuries predominating in tennis.[1, 3, 6, 7] A study by Hutchison et al [8], which followed 1440 tennis players over a 6 year period, showed that the lower extremity provided the majority of sprain type injuries with 87,5% of ligament sprains coming from the knee and ankle.

To our knowledge, in the current literature there are no studies regarding the movement patterns of the lower extremities during tennis. However, when one observes the lower limb of the players during tennis, a forward lunge movement is one of the most frequent exerted movements during the game. Because of the load these movements place on the knee joint, problems in the knee are related to the constant pounding that occurs during play.[9, 10] Therefore, as forward lunges are performed very frequently during tennis, it is of great importance that the player exerts this movement in a correct physiological way with respect to the knee joint to prevent injury. This implicates that during the forward lunge the movement of the knee in the frontal plane is to be kept within “its physiological limits” i.e. a Q-angle not exceeding the purported pathological limit of 15-20° [12].

The Quadriceps (Q) angle is a clinical measure of the alignment of the quadriceps femoris musculature relative to the underlying skeletal structures of the pelvis, femur and tibia.[11] This angle is formed by the imaginary line from the anterior superior iliac spine to the centre of the patella and from the centre of the patella to the middle of the anterior tibial tuberosity. It has been stated that when the Q-angle exceeds 15-20°, it is commonly thought to contribute to knee extensor dysfunction and hence knee injuries such as patellofemoral pain.[12]

In the literature it is an accepted fact that proximal core hip strength is needed for control of distal segments.[13] Therefore the force of the muscles surrounding the hip may play an important role in controlling the movement of the knee in the frontal and transversal plane. Ireland et al [14] postulated that uncontrolled femoral adduction and internal rotation secondary to hip weakness results in an increase in the dynamic Q-angle at the knee.

Repetitive activities with this malalignment may eventually lead to knee injury. Hence, hip muscle weakness might be associated with impaired biomechanics and postures of the leg that contribute to lower extremity injuries.[15] It is therefore currently targeted as one of the possible predisposing factors for knee overuse injuries such as anterior knee pain and iliotibial band friction syndrome and lower leg injuries such as medial tibial stress syndrome.[13, 14, 16, 17]

Although there is a reason to assume that hip muscle weakness could effect the frontal plane movement of the knee during lunge movements in tennis, to our knowledge, no studies have been published which have investigated the relationship between hip muscle strength and the movement pattern of the knee in the frontal plane during this manoeuvre. Therefore, the purpose of this study was to test the hypothesis that the strength of the muscles around the hip are related to the frontal plane motion of the knee during a functional lunge movement.

## **MATERIALS AND METHODS**

### **Subjects**

84 officer cadets (76 men, 8 women) of the Belgian Royal Military Academy, who were without a history of any hip, knee or lower leg complaints were recruited for this study. The average age of the subjects was 19,2 years (range, 18 to 30). The cadets had an average height of 177, 7 cm (range 160,0 to 192,0) and an average weight of 70,2 kg (range 42 to 91). The aim of the study was explained to each subject and they all signed an informed consent. The study was approved by the Ethical Commission of Belgian Defence. Before testing, all cadets visited the same sports physician for a comprehensive injury history and a clinical examination of the knee joint. Subjects who had a history of a surgical procedure involving the hip, knee, lower leg, ankle or foot or a history of an injury to the hip, knee, lower leg, ankle or foot within six months before the start of the study were excluded from the study.

### **Evaluation**

The muscle strength of the 6 major muscle groups of subject's both hips was measured using a hand-held dynamometer. The frontal plane movement of the subject's knees during a forward lunge was recorded using a Sony HC20E camera which was placed in front of the subject perpendicular to the frontal plane.

## **Muscle strength testing**

Strength testing of the hip muscles was performed using a Microfet hand-held dynamometer (Hoggan Health Industries, West Jordan, UT). The test-retest reliability of muscle testing in the lower extremity using a hand-held dynamometer has shown intraclass correlation coefficient values of 0.95 to 0.99 [18], 0.68 to 0.79 [19] and 0.74 to 0.80 [20]. The 6 major muscle groups of the subject's both hips were tested in a randomly determined order. The tested muscles were: hip flexors, extensors, abductors, adductors, internal and external rotators. During the test procedure the subject applied a maximum isometric muscle contraction to the examiner's hand, holding the dynamometer in a fixed position (make-method). After a practice trial, three trials were performed. The muscle contraction was held for 5 seconds with 15 seconds of rest between trials.

Muscle testing was performed in consistency with the methods of muscle testing described by Reese.[21] During the test the subjects were instructed to hold their arms crossed over their chest to prevent them from self stabilizing by holding their hands on the table. Hip flexion was tested in a seated position. Resistance was applied with the dynamometer placed on the anterior aspect of the distal thigh at 2cm proximal to the knee. Hip extension was tested in a prone position with resistance applied on the posterior aspect of the distal thigh at 2cm proximal to the popliteal crease. Hip abduction was tested supine with the hip of the limb to be tested abducted and in neutral position with the knee extended. Resistance was applied on the lateral aspect of the distal thigh at 2cm proximal to the lateral epicondyle of the knee. Hip adduction was also tested supine with the non tested limb in full abduction, the test limb in adduction and the knees extended. Resistance was applied at 2cm proximal to the medial epicondyle of the knee. Internal and external hip rotation were tested in a seated position with resistance applied 2cm proximal to respectively the lateral and medial malleolus.

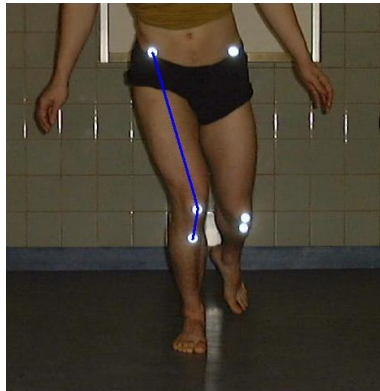
The peak force from the 3 trials was used for data analysis. In addition, flexion/extension (Flex/Ext), abduction/adduction (Abd/Add) and external rotation/internal rotation (ER/IR) force ratios were calculated and used for analysis. Prior to data analysis, strength measurements, recorded in Newton, were normalized to body weight for each subject.

## **Evaluation of knee frontal plane movement**

Subjects were videotaped as they performed a series of forward lunges (Fig.1). A Sony HC20E camera was positioned on a 60 cm high stand in front of the subject, perpendicular to the frontal plane of the knee at a distance of 2,5 meters from the subject. In this way two

dimensional video data of the frontal plane movement of the knee were collected. For both legs the subjects were asked to perform a series of three forward lunges starting from a standing position with both feet at shoulder width. The subjects performed the lunge movement barefoot. The knee flexion angle of the weightbearing extremity during the lunge movement was limited to  $45^\circ$  by varying the distance over which the lunge had to be performed.

To determine the knee valgus/varus angles for each subject reflective markers were fixed to the skin over anatomical landmarks. Markers were placed on the right and left anterior superior iliac spines, the centre of the left and right patella and the middle of the left and right anterior tibial tuberosity. A digital video analysis software program, Darttrainer 2.5 (Dartfish video software solutions, Fribourg, Switzerland), was used to determine the peak knee valgus or varus angles for each subject during the three forward lunge movements with each leg. The average valgus or varus angle of the left and right leg was calculated and used for statistical analysis.



**Figure 1:** Frontal plane posture of a subject's knee during a forward lunge.

### **Statistical analysis**

Based on the video-analysis of the frontal plane movement of the knees during the recorded forward lunge movements, subjects could be divided into two groups: a group which moved their knee into valgus (valgus group) and a group which moved their knee into varus (varus group) during forward lunge.

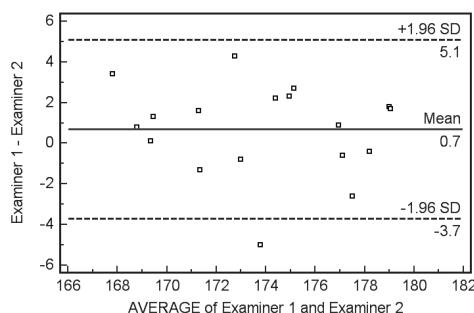
Statistical analysis was performed with SPSS for windows version 12.0 (SPSS Inc, Chicago, Ill). To assess the repeatability of the digital video analysis of the peak knee valgus and varus



angles during the lunge movement, the Bland-Altman plot was used to show the range of agreement between the first and the second examiner. [22] In the graph the difference between the two examiners' scores was plotted against the average of the measurements. The Kolmogorov – Smirnov test was used in order to indicate a normal distribution of the data. For the tested six major muscle groups, Independent-Samples T-tests were used to compare hip muscle strength differences between the valgus group and the varus group. In both groups the Pearson product-moment correlation coefficient was used to examine the relationship between the force and agonist/antagonist force ratios of the hip muscles and the amount of knee movement in the frontal plane during a forward lunge. Statistical significance was accepted at the level of  $\alpha \leq 0.05$ .

## RESULTS

The Bland-Altman plot (Fig. 2) shows that the differences within mean  $\pm 1.96$  SD ( $0.69 \pm 4.39$ ) are not clinically relevant, confirming a good repeatability of the score of the digital video analysis of the peak knee valgus and varus angles as 95% of the differences were less than two SD.



**Figure 2:** Bland-Altman plot difference between two examiner's scores against average of the digital video analysis scores of the peak knee valgus and varus angles during the lunge movement.

There were no significant differences in muscle strength for none of the 6 tested muscle groups between the valgus group and the varus group (table 1).

**Table 1:** Mean, standard deviation (SD) and P-values of the t-test of the strength (in Newton, normalized to body weight) of the 6 tested hip muscle groups of the valgus and varus group.

Hip muscle strength (N)	Mean valgus group	SD valgus group	Mean varus group	SD varus group	Significance t-test
Flexor	389.08	68.16	375.48	57.25	0.41
Extensor	559.11	152.86	571.52	116.50	0.73
Abductor	371.26	57.41	391.79	38.98	0.12
Adductor	377.38	79.83	381.62	61.63	0.82
Internal rotator	238.23	42.87	241.04	38.40	0.79
External rotator	244.88	48.69	254.47	33.39	0.39

( $\alpha = 0.05$ )

Neither in the valgus group nor in the varus group, a significant correlation could be revealed between the strength of any of the assessed muscle groups and the amount of movement into valgus/varus (table 2).

**Table 2:** Correlation coefficients (r) and P-values of the correlations between the strength of the hip muscle groups (in Newton) and the amount of movement into valgus/varus.

Hip muscle strength (N)	Valgus group		Varus group	
	Corr. Coeff. (r)	p-value	Corr. Coeff. (r)	P-value
Flexor	0.19	0.23	0.03	0.89
Extensor	0.11	0.49	- 0.20	0.35
Abductor	- 0.002	0.99	- 0.41	0.06
Adductor	0.03	0.84	- 0.32	0.12
Internal rotator	- 0.22	0.15	- 0.39	0.06
External rotator	- 0.05	0.75	0.21	0.33

( $\alpha = 0.05$ )

In the valgus group no significant correlation was found between the force ratios of the hip muscles, Flex/Ext ( $r = - 0.08$ ,  $P = 0.40$ ), Abd/Add ( $r = - 0.15$ ,  $P = 0.12$ ) and ER/IR ( $r = 0.14$ ,  $P = 0.15$ ), and the amount of valgus movement at the knee. However, in the varus group the ER/IR force ratio was positively related to the amount of knee varus during the forward lunge movement ( $r = 0.31$ ,  $P = 0.03$ ).

No statistical significant differences could be detected for the results of the assessed strength of the 6 tested hip muscle groups, the agonist/antagonist force ratios or the amount of knee movement in the frontal plane during the forward lunge between the male and female subjects.

## DISCUSSION

It has been demonstrated that muscle weakness proximal to a symptomatic area is often present in lower-extremity injury conditions.[14, 16, 17, 23-25] The closed kinetic chain theory suggests that sufficient proximal hip strength is needed for control of distal segments to prevent injury. If a joint of the lower extremity is not functioning properly, injuries can be manifested in other joints, particularly those that are distal to the affected joint. It is therefore speculated that hip muscle weakness may play a role in knee overuse injuries.[13]

In this current study no significant strength differences of the 6 major muscle groups around the hip were found between the group of subjects which knee moved into valgus and the group which knee moved into varus during a forward lunge movement. In addition, in none of both groups a significant relationship was found between the strength of the assessed hip muscles and the amount of knee valgus/varus movement during the lunge. Therefore, the idea that the strength of the muscles around the hip is related to the amount of knee movement into valgus or varus during movements such as a forward lunge may not be valid in healthy individuals.

As previously stated by Ireland et al [14], to date, studies that have investigated the relationship between lower-extremity frontal plane stability and the prevention of knee injuries are scarce. A study by Hewett et al [26] demonstrated that following a 6-week training program including lower-extremity plyometric drills and general strength and flexibility exercises, there was a 50% reduction in the adduction/abduction moments at the knee during the landing phase of a vertical jump. Although the training program did not focus on the hip muscles specifically, the decrease in frontal plane knee moment was the only significant predictor of the athlete's risk for knee ligamentous injury. A subsequent prospective study by the same authors showed that female athletes who participated in the same training program had a significantly lower incidence of severe knee ligament injury than those who did not.[27]

In this current study, except for the ER/IR force ratio in the varus group, no relationship could be established between hip muscle strength and frontal plane movement of the knee during a tennis lunge. However in distance runners who presented with iliotibial band syndrome, Fredericson et al [28] postulated that strengthening of the gluteus medius fostered increased control of thigh adduction and internal rotation tendencies during running, thereby

minimizing the valgus vector at the knee. Also Ireland et al [14] indicated that strengthening of the abductors and external rotators of the hip may benefit individuals suffering from anterior knee pain by improving stability of the lower extremity in the frontal and transverse planes of motion during sport-specific activities.

In this current study, however, we did find that in the group of subjects which knee moved into varus the ER/IR force ratio at the hip was moderately, yet significantly positively related to the amount of knee varus during a forward lunge movement. This indicates that when the external rotator muscle strength exceeds the muscle strength of the internal rotators of the hip, the knee is moved more into varus during this particular movement. The positive correlation between the ER/IR force ratio and the degree of varus movement during the lunge movement, found in this study, may concur with the findings of Fredericson et al in a way that a stronger force of the external rotators of the hip relative to the internal rotator force may cause the knee to be moved more laterally during activities such as a forward lunge.

One possible explanation for the fact that in our study further no correlation between hip muscle strength and the amount of varus/valgus movement at the knee during the forward lunge could be established could be that the tested population consisted of all healthy subjects without any hip, knee or lower leg complaints at the moment of the study. It is plausible that a relationship between the investigated parameters could manifest itself in a population suffering from knee or other lower extremity injuries and presenting hip muscle weakness.

In addition, fatigue of the hip muscles could be a factor which may have an effect on the relationship between the strength of the hip muscles and the amount of knee varus/valgus during forward lunge movements in tennis. It may be possible that this relationship only reveals itself when the muscles are getting fatigued during prolonged tennis playing. As the duration of tennis matches is usually 90-120 minutes on grass and fast surfaces and 120-180 minutes on clay, an important subject is the time course of changes in muscle strength during this prolonged intermittent exercise.[29] Girard et al [29] found in well trained male tennis players that a progressive reduction in maximal voluntary strength of the knee extensors was highly correlated with increases in perceived exertion throughout a three hour tennis match. If this is also the case for the hip musculature, this could possibly have an effect on the movement patterns of the knee in the frontal and transversal plane. However this condition was not assessed in this study.

However, in the investigated population of this study the strength of the hip muscles did not seem to be related to the degree of knee valgus and varus during the forward lunge. As postulated earlier, this could also mean that, in an asymptomatic population, hip muscle strength may not be such an important factor in the control of the frontal plane knee posture during this kind of movement as has been hypothesized in the beginning of this study. This may suppose that other factors, such as good proprioception and sufficient core hip stability, instead of the absolute strength of the hip muscles could be more important in the control of knee movement during a forward lunge movement. Proprioception is granted to be of great importance in the coordination of skill-demanding movements which are performed in tennis and therefore also contributes to lower limb control during tennis playing.[30] As it has been predicted that tennis players' proprioception would be directly related to the amount of practice [30], based upon the results of this study, it may be assumed that proprioceptive training of the lower extremities may be more appropriate than purely strength training of the hip muscles to ensure the quality of a tennis-specific movement such as a forward lunge.

No significant differences could be found between the male and female subjects, neither for the assessed strength of the 6 tested hip muscle groups and the agonist/antagonist force ratios, nor for the amount of knee movement in the frontal plane during the forward lunge. However, we believe that in this study the number of female subjects (n = 8) may be too small to detect possible differences between males and females in this regard.

A limitation of this study is that only healthy subjects without self reported lower extremity problems were assessed, whereas possible relationships between hip muscle strength and frontal plane movement of the knee during sport-specific activities may more likely to be found in a patient population suffering from lower limb injuries. However, it was the primary goal of this study to search for a possible relationship between hip muscle strength and the frontal plane posture of the knee during a forward lunge in an asymptomatic population. Future research should assess whether this relationship can be established in a patient population suffering from lower extremity injuries in which hip muscle weakness may be present. In addition, the question if hip muscle fatigue, which may be caused by prolonged tennis playing, has an effect on knee frontal plane movement during tennis-specific lower limb movements should be addressed in future studies.

In this study the isometric strength of the hip muscles was tested using a hand-held dynamometer. Hand-held dynamometry for measuring hip muscle strength was chosen since this method has been proven to be a reliable method. [18, 19, 20]

However, during a forward lunge there are not solely isometric contractions. Consequently, it may be more functional to examine the relationship between the frontal plane posture of the knee during a forward lunge and the isokinetic (concentric and eccentric) strength of the hip muscles. This should be addressed in future research.

## **CONCLUSIONS**

In this study no significant differences in hip strength could be detected between the subjects whose knee moved into valgus and those whose knee moved into varus during a forward lunge movement. However, in the subjects whose knee moved into varus, ER/IR force ratio was moderately related to the amount of knee varus during the lunge movement, the results of this study suggest that in healthy individuals hip muscle strength is not correlated to the amount of valgus/varus movement of the knee during a forward lunge. This does suspect that instead of the absolute strength of the hip muscles other factors such as sufficient proprioception and core hip stability may be more important for controlling knee movement during this tennis-specific movement.

## REFERENCES

1. Mohtadi N, Poole A. Racquet Sports. In: Caine DJ, Caine CG, Lindner KJ, eds. *Epidemiology of sports injuries*. Champaign, Illinois: Human Kinetics Publishers 1996: 301-311.
2. Maquirriain J, Ghisi JP. The incidence and distribution of stress fractures in elite tennis players. *Br J Sports Med* 2006; 40: 454-459.
3. Pluim BM, Staal JB, Windler GE, *et al.* Tennis Injuries: occurrence, aetiology, and prevention. *Br J Sports Med* 2006; 40: 415-423.
4. Berson BL, Rolnick AM, Ramos CG, *et al.* An epidemiologic study of squash injuries. *Am J Sports Med* 1981; 9(2): 103-106.
5. Soderstrom CA, Doxanas MT. Racquetball: A game with preventable injuries. *Am J of Sports Med* 1982; 10(3): 180-183.
6. Chard MD, Lachman SM. Racquet sport patterns of injury presenting to a sports injury clinic. *Brit J Sports Med* 1987; 27(4): 150-53.
7. Barber FA, Sutker AN. Iliotibial band syndrome. *Sports Med* 1992; 14(2): 144-48.
8. Hutchinson MR, Laprade RF, Burnett QM, *et al.* Injury surveillance at the USTA boys tennis championships – A 6-year study. *Med Sci Sports Exerc* 1995; 27(6): 826-30.
9. Gecha SR, Torg E. Knee injuries in tennis. *Clin Sports Med* 1988; 7(2): 435-52.
10. Leach RE. Leg and foot injuries in racquet sports. *Clin Sports Med* 1988; 7(2): 359-70.
11. Livingston LA, Spaulding SJ. OPTOTRAK measurement of the Quadriceps angle using standardized foot positions. *Journal of Athletic Training* 2002; 37(3): 252-55.
12. Livingston LA. The Quadriceps Angle: A review of the literature. *J Orthop Sports Phys Ther* 1998; 28(2): 105-109.
13. Niemuth PE, Johnson RJ, Myers MJ, *et al.* Hip muscle weakness and overuse injuries in recreational runners. *Clin J Sport Med* 2005; 15(1): 14-21.
14. Ireland ML, Willson JD, Ballantyne BT, *et al.* Hip strength in females with and without patellofemoral pain. *J Orthop Sports Phys Ther* 2003; 33(11): 671-76.
15. Hollman JH, Kolbeck KE, Hitchcock JL, *et al.* Correlations between hip strength and static foot and knee posture. *J Sports Rehab* 2006; 15(1): 12-23.
16. Tyler TF, Nicholas SJ, Mullaney MJ, *et al.* The role of hip muscle function in the treatment of patellofemoral pain syndrome. *Am J of Sports Med* 2006; 34(4): 1-7.
17. Mascal CL, Landel R, Powers C. Management of patellofemoral pain targeting hip, pelvis and trunk muscle function: 2 case reports. *J Orthop Sports Phys Ther* 2003; 33(11): 647-60.

18. Wang CY, Olson SL, Protas EJ. Test-retest strength reliability: hand-held dynamometry in community-dwelling elderly fallers. *Arch Phys Med Rehab* 2002; 83: 811-15.
19. Kimura IF, Jefferson LM, Gulick DT, *et al.* Intra- and intertester reliability of Chatillon and Microfet hand- held dynamometers in measuring force production. *J Sport Rehab* 1996; 5: 197-205.
20. Agre JC, Magness JL, Hull SZ, *et al.* Strength testing with a portable dynamometer: reliability for the upper and lower extremities. *Arch Phys Med Rehab* 1987; 68: 454-58.
21. Soderberg GL. Handheld dynamometry for muscle testing. In: Reese NB. *Muscle and sensory testing*. Philadelphia, Pennsylvania: WB Saunders Company 1999: 405-10.
22. Bland JM, Altman DG, Statistical methods for assessing agreement between two methods of clinical measurement. *Lancet* 1986; 1: 307-10.
23. Nicholas JA, Strizak AM, Veras G. A study of thigh muscle weakness in different pathological states of the lower extremity. *Am J Sports Med* 1976; 4: 241-48.
24. Jaramillo J, Worrell TW, Ingersoll CD. Hip isometric strength following knee surgery. *J Orthop Sports Phys Ther* 1994; 20: 160-65.
25. Janda V. Muscle strength in relation to muscle length, pain and muscle imbalance. In: Harms-Ringdahl K, ed. *Muscle strength (International Perspectives in Physical Therapy)*. Edinburgh, UK: Churchill Livingstone 1993: 83-91.
26. Hewett TE, Stroupe AL, Nance TA, *et al.* Plyometric training in female athletes. Decreased impact forces and increased hamstring torques. *Am J Sports Med* 1996; 24: 765-73.
27. Hewett TE, Lindenfeld TN, Riccobene JV, *et al.* The effect of neuromuscular training on the incidence of knee injury in female athletes. A prospective study. *Am J Sports Med* 1999; 27: 699-706.
28. Fredericson M, Cookingham CL, Chaudhari AM, *et al.* Hip abductor weakness in distance runners with iliotibial band syndrome. *Clin J Sport Med* 2000; 10: 169-75.
29. Girard O, Lattier G, Micallef JP, *et al.* Changes in exercise characteristics, maximal voluntary contraction, and explosive strength during prolonged tennis playing. *Br J Sports Med* 2006; 40: 521-526.
30. Lin CH, Lien YH, Wang SF, *et al.* Hip and knee proprioception in elite, amateur, and novice tennis players. *Am J Phys Med Rehabil* 2006; 85: 216-21.



## **CHAPTER 5**

### **Does bracing influence brain activity during knee movement: An fMRI study**

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## Abstract

**Background:** Studies have shown that proprioceptive input during active and passive arm movements is processed in the primary and secondary somatosensory cortex and supplementary motor area of the brain. At which level of the central nervous system proprioceptive signals coming from the knee are regulated remains to be elucidated.

**Purpose:** In order to investigate if there is a detectable difference in brain activity when various proprioceptive signals are exerted at the knee, functional magnetic resonance imaging (fMRI) was used.

**Study Design:** Crossover Study Design.

**Methods:** fMRI in thirteen healthy, right leg dominant female volunteers compared brain activation during the performance of flexion-extension movements with the right knee under three different conditions: with application of a tight knee brace, with application of a moderate tight knee sleeve, and without application of a brace or sleeve.

**Results:** Brain activation was detected in the primary sensorimotor cortex (left and right paracentral lobule) and the left superior parietal lobule of the brain. There was a significantly higher level of brain activation with the application of the brace and sleeve, respectively, compared to the condition without a brace or sleeve. A significantly higher cortical activation was also seen when comparing the braced condition with the condition when a sleeve was applied.

**Conclusions:** The results suggest that peripheral proprioceptive input to the knee joint by means of a brace or sleeve seems to influence brain activity during knee movement. It appears that the intensity of brain activation during knee movement can be influenced by the intensity of proprioceptive stimulation at the joint.

**Clinical Relevance:** By stimulating the cortical areas responsible for the processing of kinaesthetic signals, bracing may simultaneously stimulate the areas which are involved in the generation of motor activity and in this way enhance motor control.

**Key Terms:** Brain activation; proprioception; knee movement; brace

## INTRODUCTION

Functional magnetic resonance imaging (fMRI) studies have recently shown that isolated lower limb movements require the activation of a distributed motor network in the human brain including the primary and non-primary motor and sensory areas, such as the sensorimotor cortex (SMC), premotor cortex (PMC), supplementary motor area (SMA), cingulate motor area (CMA), secondary sensory cortex (SII), basal ganglia, thalamus and cerebellum (14,15). These studies have indicated that the cortical representations of the knee, ankle and toes show a somatotopic organization which is in accordance with the orientation of the classic homunculus as described by Penfield and Rasmussen (18).

It has been shown that the primary and secondary somatosensory cortex, as well as the supplementary motor area are involved in the central processing of proprioceptive signals during passive and active arm movements (21). However, what the contributions of the different cortical areas are for the processing of proprioceptive information, coming from the lower limb joints has not yet been elucidated.

Proprioception, the ability to perceive one's own body movements and limb positions in space, describes afferent information arising from various mechanoreceptors (Ruffini endings, Pacinian corpuscles and Golgi organs), nociceptors (bare nerve endings) and muscle afferents (spindles and Golgi tendon organs) that contribute to postural control, limb movement, joint stability and several conscious sensations (12,22,27). Clinical experience indicates that good proprioceptive abilities play a major role in protecting a joint, such as the knee, against injury (10).

Research has shown that braces and elastic bandages improve knee joint proprioception. (2,4,16,19). Knee braces are therefore frequently used in the treatment and prevention of many common knee problems in sports and clinical settings. Many mechanisms have been suggested for the effects of braces (2,13,20,28). Researchers hypothesise that bracing enhances proprioception by increasing cutaneous stimuli and pressure on the underlying musculature and capsule of the joint that it surrounds (19,24). Edin (8) has demonstrated that cutaneous mechanoreceptors provide high-fidelity information about knee joint movements. He states that the stabilizing effects of bracing of large joints such as the knee are attributable to the altered somatosensory inflow from the skin. However, although there's objective evidence in the literature that wearing a knee brace improves the proprioception of the joint, the mechanism by which bracing seems to influence proprioception remains enigmatic. In the literature there is controversy concerning at which level of the central nervous system (CNS) proprioception is regulated. It has been suggested that improvements in proprioception, as a

result of wearing a brace, may indicate that braces and sleeves provide additional somatosensory cues that reflexively bias proprioceptive pathways (3,28). Other authors suggest however that proprioception is conveyed to all levels of the central nervous system and the majority of sensory inputs from the mechanoreceptors are processed through the dorsal root spinal ganglion, ascend through the posterior spinal cord and are conducted to the cerebral cortex (13,23). Consequently, the question arises whether alterations in proprioceptive input are regulated through reflexive pathways or by higher motor control centres. Therefore, the purpose of this study was to investigate if there is a detectable difference in brain activity during movement of the lower limb under three different conditions: wearing a tight knee brace, wearing a moderate tight knee sleeve, and without wearing a brace or a sleeve. We hypothesised that a higher activation pattern would be present in the higher described primary and non-primary motor and sensory areas of the brain when comparing the braced versus the non braced condition.

## **MATERIALS AND METHODS**

### **Subjects**

Thirteen right leg dominant female volunteers (aged  $19 \pm 1,5$  years; range 18-23 years) participated in the study after giving a written informed consent. Before the testing procedure, all subjects underwent a comprehensive verbal screening to make sure that they did not met any of the exclusion criteria for the fMRI study. These exclusion criteria were: A history of neurological or cardiovascular disease, medications, cardiac or neural pacemakers, metal objects in the body, and a history of musculoskeletal injury in both lower limbs. The study was approved by the ethical committee of the Ghent University Hospital.

### **Experimental design**

Subjects were positioned head first and supine inside the MRI-scanner till the level of the pelvis. The foot of the subject's right leg rested on a custom-built wooden platform which contained a groove in which the subject's foot could slide back and forth during the flexion-extension movement of the knee. The groove in the wooden platform was provided with an adjustable stop which limited the flexion-extension movement of the knee from 0 to 90 degrees. Subjects performed unilateral flexion-extension movements of the knee under three different conditions: 1) with a tight brace around the knee, 2) with a moderate tight sleeve

surrounding the knee, and 3) without wearing a brace or sleeve. Each movement condition lasted 30 s and was triggered by a visual command (Presentation, [www.neurobs.com](http://www.neurobs.com)). In addition, to visually pace the movements, Presentation® was used to impose a constant timing and equal number of cycles across the conditions. In between the periods of knee movement there was a resting period of 20 s during which the brace or sleeve was putted on or removed by an assistant researcher, who was standing outside the scanner. To restrain head motion, subjects were fixed with straps around the forehead and a bite-bar device as described by Kapreli et al. (14,15). In addition, a strap was placed over the subject's hips to further limit head motion as a consequence of lower limb motion (Figure 1).



**Fig.1** (A) Positioning of the subject, showing the custom-built wooden platform, head motion restriction precautions (the bite bar and strap over the hips) and tight brace around the knee. (B) Same positioning, with a moderate tight sleeve around the knee.

### **fMRI data acquisition**

Imaging was performed on a 3 T MR scanner (MAGNETOM Trio, Siemens AG, Erlangen, Germany) using an eight-channel head coil for radio frequency transmission and signal reception. Each scanning session consisted of 475 whole brain gradient-echo echoplanar scans (EPI), which were acquired every 2.5 s, with anterior-posterior encoding direction (TR/TE = 2500/33 ms, field of view = 192 mm, matrix = 64 x 64, slice thickness = 3mm + 1 mm interslice distance, 33 sagittal slices), followed by an anatomical 3D high-resolution T1-weighted image (MPRAGE) (TR/TE = 15550/2.39 ms, field of view = 220 mm, matrix = 256 x 256, slice thickness = 0.9 mm, 176 slices).

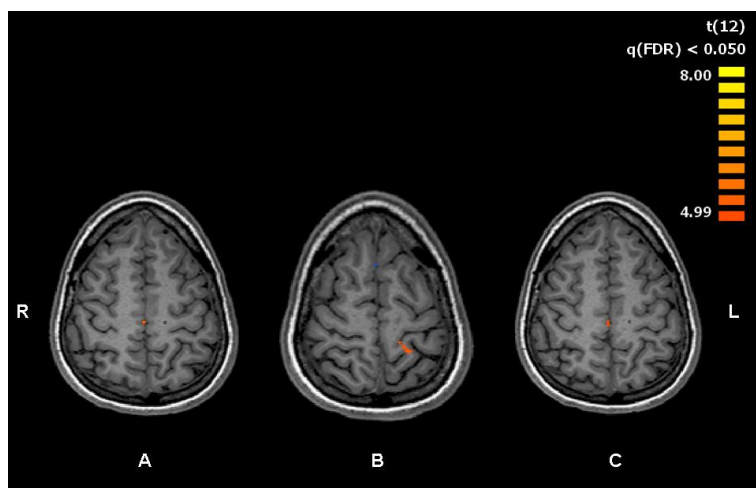
## Data analysis

fMRI data were statistically analyzed using Brainvoyager QX Version 1.9 (Brain Innovation, Maastricht, The Netherlands) (11). The functional dataset acquired from each paradigm consisted of 475 image volumes. Functional data were converted into Brain Voyager's FMR format and subjected to a standard sequence of preprocessing steps comprising slice scan time correction using sinc interpolation, 3-D motion correction by spatial alignment to the first volume using sinc interpolation, and temporal filtering using linear trend removal and high pass filtering for low-frequency drifts of 3 or fewer cycles per time point. Estimated rotation and translation parameters before head motion correction never exceeded 3 mm and were corrected adequately by the spatial alignment. Spatial smoothing using a Gaussian filter (FWHM = 8 mm) was applied for the volume based analysis. Functional echo-planar imaging sequences were co-registered with the subject's 3-D anatomical dataset. Then, they were transformed into Talairach space (25), resulting in normalized 4-D volume time course (VTC) data. A general linear model (GLM) multistudy analysis was undertaken. Throughout the study a threshold of  $P < .05$  corrected for multiple comparisons using False Discovery Rate (FDR) correction was consistently applied. The activated regions of the averaged multistudy of all subjects in the Talairach brain were identified using the Talairach (25) and customary anatomical atlases.

## RESULTS

Evaluation by a general linear model multi-subject analysis showed a significant cortical activation during the flexion-extension movements of the knee under all three conditions (brace, sleeve, no brace or sleeve). As expected, the left cerebral hemisphere contralateral to the body side of movement was predominantly activated. In accordance with the orientation of the classic homunculus, brain activity during lower limb movement was located in the primary sensorimotor cortex (SM1) in the paracentral lobule and in the superior parietal lobule of the brain.

Random effects GLM corrected for temporal serial correlation and multiple comparisons (FDR) revealed a significantly higher level of activation in the SM1 in the paracentral lobule of the right frontal lobe (Brodmann area 5) when flexion-extension movement of the knee with the application of a knee brace was compared to the movement of the knee without a brace or sleeve (fig.2A).



**Fig. 2** Cerebral activation during flexion-extension movement of the right knee (A = brace > no brace/sleeve, B = sleeve > no brace/sleeve, C = brace > sleeve).

Knee movement while wearing a sleeve around the knee generated significantly more activation in the superior parietal lobule of the left parietal lobe (Brodmann area 5) in comparison with the no brace or sleeve condition (fig.2B).

An increased activation in the SM1 in the paracentral lobule of the left frontal lobe (Brodmann area 5) could also be observed when flexion-extension movement of the knee with the application of the tight brace was compared to the movement of the knee when a moderate tight sleeve was applied (fig.2C).

The results of the analysis comparing the activated brain areas during right knee flexion-extension movements while wearing a knee brace, a knee sleeve, and without wearing a brace or sleeve, respectively, are summarized in table 1.

**Table 1:** Brain activation when comparing the movement of the knee while wearing a knee brace, a knee sleeve, and without wearing a brace or sleeve.

Task	Condition	Brain region	BA	Talairach coordinates			Cluster size	t <sub>max</sub>
				x	y	z		
Flex-ext movement right knee	Brace vs no brace	Frontal lobe, paracentral lobule	5	1	-28	53	23	5.84
	Sleeve vs no brace	Parietal lobe, superior parietal lobule	5	-19	-43	61	22	5.64
	Brace vs sleeve	Frontal lobe, paracentral lobule	5	0	-30	53	17	4.78

(BA = Brodmann area)

DISCUSSION

To our knowledge, the present study is the first to investigate the influence of different proprioceptive signals at the knee joint (by means of the application of a knee brace and sleeve) on the cortical activity during movement of the knee.

We found that flexion and extension movement of the right knee was represented by cortical activation in the SM1 in the paracentral lobule and in the superior parietal lobule of the left hemisphere. This finding is in accordance with the results of a study by Kapreli et al. (14) and is in agreement with the classic homunculus (18).

Striking in this study was that during knee movement subjects exhibited a significant higher level of brain activation when a tight brace was applied around the knee compared to the condition when no brace was present at the knee. In addition, a significantly higher activation of the brain during flexion-extension movement of the knee was also seen when a moderately tight sleeve was applied around the knee. Although it has been demonstrated that knee joint proprioception improves with the application of a knee brace, today it is still uncertain in the literature what mechanism is responsible for the improvement in knee proprioception (2,5,13,16,19,26,27). It has been suggested by some researchers that improvements in knee joint proprioception, as a result of the application of a knee brace, are caused by additional somatosensory cues that are processed through reflex loop mechanisms (3,28). Others state



however that proprioceptive signals coming from mechanoreceptors in the skin, joint capsule, ligaments and muscles are conducted to higher levels of the central nervous system and are processed in the cerebral cortex (13,23). It has been demonstrated that the sensory afferents from muscle spindles, cutaneous, and joint receptors contribute to the signalling of limb movements to the brain, and the brain processes these sensory inputs to create perceptual representations of limb movements (6,7,8,9). Yet, little is known about where within the CNS the processing of different types of proprioceptive inputs, at a specific joint, is situated. A study by Radovanovic et al. (21) has demonstrated that proprioceptive information coming from the upper limbs are centrally processed in the primary and secondary somatosensory cortex and in the supplementary motor area of the brain. However, with respect to the lower limbs it is not clear whether different cortical areas are involved in the processing of different proprioceptive inputs and what their relative contributions might be. The results of this present study indicate that varying proprioceptive inputs at the knee, as a result of cutaneous stimulation and increased pressure on the underlying musculature and capsule of the joint by the application of a brace or sleeve, influences brain activity in the primary sensorimotor cortex and is not only processed by a reflex loop mechanism.

Interestingly, a significantly higher cortical activation could also be observed during knee movement while the subjects were wearing the tight brace around the knee compared to the movement of the knee with the application of the moderately tight sleeve. This may indicate that the intensity of proprioceptive stimulation influences the degree to which the sensorimotor cortex is activated during knee movement. Taken together, the results of this study indicate that the primary sensorimotor cortex is involved in the central processing of proprioceptive signals during active movements of the knee and that the degree to which this brain area is activated can be influenced by varying the somatosensory inflow from the skin. The findings of this study are in line with the general opinion about the role of the primary motor and somatosensory area in processing of proprioceptive input and in the generation of the movement sense (21).

The section of the brain which was active during knee movement when a knee brace (Talairach coord: -7, -27, 61) and sleeve (Talairach coord: -8, -26, 62) were applied respectively, corresponded well to the section which was activated during knee movement without brace or sleeve application (Talairach coord: -2, -27, 64). This may indicate that identical sections of motor areas are engaged both in kinaesthetic sensory processing and in the generation of corresponding limb movement. Consequently, it is possible that the cortical

areas that generate motor activity also process kinaesthetic signals related to the same movement. The tight coupling between the peripheral afferent input reaching the somatosensory cortex and the motor output in the motor areas is important for understanding how peripheral proprioceptive input can influence the motor commands and execution of movements.

This points out important clinical implications for the use of bracing in the prevention of knee injuries. It has been indicated that the proprioceptive input caused by cutaneous stimulation as a result of taping and bracing of the knee joint has an impact on the recruitment of the muscles surrounding the joint (17,26). By stimulating the cortical areas responsible for the processing of kinaesthetic signals, bracing may simultaneously stimulate the areas which are involved in the generation of motor activity and in this way enhance motor control. By improving muscle recruitment and motor control of a joint in this way, bracing may especially be important for subjects suffering from functional instability of the knee due to poor proprioceptive acuity.

## **CONCLUSIONS**

This study shows that different proprioceptive inputs to the knee joint by means of a brace or sleeve seem to have a direct influence on brain activity during knee movement. An increased level of brain activation was seen with the application of a brace and sleeve, respectively, compared to the condition when no brace or sleeve was present at the knee. In addition, the higher cortical activation during knee movement with the application of a tight brace compared to the application of a moderate tight sleeve suggests that the intensity of brain activation during knee movement can be influenced by the intensity of peripheral proprioceptive stimulation at the joint. Improvement of knee joint proprioception by the application of braces and elastic bandages appears to be the result of the stimulation of the primary sensorimotor cortex, which shows to be involved in central processing of proprioceptive signals coming from the knee.

## REFERENCES

1. Barrack RL, Lund PJ, Skinner HB, 1994. Knee joint proprioception revisited. *J Sport Rehab.* 3, 18-42.
2. Barret DS, Cobb AG, Bentley G, 1991. Joint proprioception in normal, osteoarthritic and replaced knees. *J Bone Joint Surg.* 73B, 53-56.
3. Birmingham TB, Inglis JT, Kramer JF, Vandervoort AA, 2000. Effect of a neoprene sleeve on knee joint kinesthesia: comparison of active, passive and axially loaded joint angle replication tests. *Med Sci Sports Exerc.* 32, 304-308.
4. Birmingham TB, Kramer JF, Inglis JT, Mooney CA, Murray LJ, Fowler PJ, Kirkley S, 1998. Effect of a neoprene knee sleeve on knee joint position sense during sitting open kinetic chain and supine closed kinetic chain tests. *Am J Sports Med.* 26, 562-566.
5. Birmingham TB, Kramer JF, Kirkley A, Inglis T, Spaulding SJ, Vandervoort AA, 2001. Knee bracing after ACL reconstruction: effects on postural control and proprioception. *Med Sci Sports Exerc.* 33 (8), 1253-1258.
6. Burke D, Gandevia SC, Macefield G, 1988. Responses to passive movement of receptors in joint, skin and muscle of the human hand. *J Physiol.* 402, 347-361.
7. Collins DF, Refshauge KM, Todd G, Gandevia SC, 2005. Cutaneous receptors contribute to kinaesthesia at the index finger, elbow, and knee. *J Neurophysiol.* 94, 1699-1706.
8. Edin B, 2001. Cutaneous afferents provide information about knee joint movements in humans. *J Physiol.* 531, 289-297.
9. Edin B, 2004. Quantitative analyses of dynamic strain sensitivity in human skin mechanoreceptors. *J. Neurophysiol.* 92, 3233-3242.
10. Fremerey RW, Lobenhoffer P, Zeichen J, Skutek M, Bosch U, Tschern H, 2000. Proprioception after rehabilitation and reconstruction in knees with deficiency of the anterior cruciate ligament: a prospective, longitudinal study. *J Bone Joint Surg Br.* 82, 6: 801-806.
11. Goebel R, 1996. Brainvoyager: A program for analyzing and visualizing functional and structural magnetic resonance data sets. *Neuroimage* 3, 604.
12. Grigg P, 1994. Peripheral neural mechanisms in proprioception. *J Sports Rehab.* 3, 3, 2-17.
13. Kaminski T, Perrin DH, 1996. Effect of prophylactic knee bracing on balance and joint position sense. *J Atl Training.* 31, 2, 131-136.

14. Kapreli E, Athanasopoulos S, Papathanasiou M, Van Hecke P, Kelekis D, Peeters R, Strimpakos N, Sunaert S, 2007. Lower limb sensorimotor network: issues of somatotopy and overlap. *Cortex*. 43, 219-232.
15. Kapreli E, Athanasopoulos S, Papathanasiou M, Van Hecke P, Strimpakos N, Gouliamos A, Peeters R, Sunaert S, 2006. Lateralization of brain activity during lower limb joints movement. An fMRI study. *NeuroImage*. 32, 1709-1721.
16. McNair PJ, Stanley SN, Strauss GR. Knee Bracing: effect on proprioception. *Arch Phys Med Rehab*. 77, 287-289.
17. Mellor R, Hodges PW, 2006. Effect of knee joint angle on motor unit synchronization. *J Orthop Res*. 24, 1420-1426.
18. Penfield W, Rasmussen T, 1950. *The cerebral cortex of man*. New York: Macmillan.
19. Perla R, Frank C, Fick G, 1995. The effect of elastic bandages on human knee proprioception in the uninjured population. *Am J Sports Med*. 23, 251-255.
20. Prymka M, Schmidt K, Jerosch J, 1998. Proprioception in patients suffering from chondrpathia patellae. *Int J Sports Med*. 19, S60.
21. Radovanovic S, Korotkov A, Ljubisavljevic M, Lyskov E, Thunberg J, Kataeva G, Danko S, Roudas M, Pakhomov S, Medvedev S, Johansson H, 2002. Comparison of brain activity during different types of proprioceptive inputs: a positron emission tomography study. *Experimental Brain Research*, 143. 3, 276-285.
22. Riemann BL, Lephart SM, 2002. The sensorimotor system, Part I: The physiologic basis of functional joint stability. *Journal of Athletic Training*. 37, 1, 71-79.
23. Riemann BL, Lephart SM, 2002. The sensorimotor system, Part II: The role of proprioception in motor control and functional joint stability. *Journal of Athletic Training*. 37, 1, 80-84.
24. Simoneau GG, Degner RM, Kramper CA, Kittleson KH, 1997. Changes in ankle joint proprioception resulting from strips of athletic tape applied over the skin. *J Atl Train*. 32, 2, 141-147.
25. Talarach J, Tournoux P, 1988. *Co-planar Stereotaxic Atlas of the Human Brain: 3-Dimensional Proportional System – an Approach to Cerebral Imaging*. Thieme: Stuttgart.
26. Van Tiggelen D, Coorevits P, Witvrouw E, 2008. The effects of a neoprene knee sleeve on subjects with a poor versus good joint position sense subjected to an isokinetic fatigue protocol. *Clin J Sport Med*. 18 (3), 259-265.

27. Van Tiggelen D, Coorevits P, Witvrouw E, 2008. The use of a neoprene knee sleeve to compensate the deficit in knee joint position sense caused by muscle fatigue. *Scand J Med Sci Sports*. 18, 62-66.
28. You SH, Granata KP, Bunker LK, 2004. Effects of circumferential ankle pressure on ankle proprioception, stiffness and postural stability: a preliminary investigation. *J Orthop Sports Phys Ther*. 34, 449-460.



## **CHAPTER 6**

### **GENERAL DISCUSSION**





## GENERAL DISCUSSION

During the past decades, a lot of research has been undertaken to investigate the mechanisms closely related to the patellofemoral joint in the initiation of the patellofemoral dysfunction syndrome with a focus on muscular and non muscular structures which directly influence patellofemoral joint mechanics. Consequently, as outlined in the general introduction, various intrinsic risk factors such as patellar and trochlear bony abnormalities, retinacular dysfunctions, quadriceps and selective VM strength deficits, neuromuscular VM/VL timing dysfunctions, and muscle flexibility disorders have already been recognized. However, malalignment of the patellofemoral mechanism is not only caused by local patellofemoral mechanics, but reflects anatomical variations throughout the entire lower extremity.(6)

The purpose of this doctoral project therefore was to look beyond the patellofemoral joint, at the lower extremity kinetic chain and to look at variables affecting lower extremity alignment distally from the PF-joint, proximally from the PF-joint and within the central nervous system in a search for other possible sources in the aetiology of PFP.

Abnormal motions of the lower and upper leg in the transverse and frontal planes are believed to have an effect on patellofemoral joint mechanics and the patellofemoral dysfunction syndrome. However, the relationship between these biomechanical abnormalities in the lower extremity and the occurrence of PFP is still obscure. The first purpose of this doctoral project was to prospectively investigate whether certain dysfunctions in the segments, situated in the kinetic chain distal and proximal of the patellofemoral joint, predispose individuals for developing patellofemoral dysfunction syndrome (**chapters 2, 3 and 4**).

Furthermore, it has been suggested that the use of braces are effective in the conservative treatment of PFP possibly by improving proprioception. This is important since it is believed that proprioceptive input contributes to the neuromuscular control of patellar tracking.(2,19,43) However, to our knowledge, no studies have been undertaken to investigate at which level of the central nervous system the processing of this additional proprioceptive input by a brace takes place. Consequently, it remains enigmatic to which degree higher control centres in the brain are involved in the regulation of proprioceptive signals coming from the knee. Therefore, in **chapter 5** of this dissertation the intention was to explore to which degree higher control centres in the cerebral cortex are activated during knee

movement when additional proprioceptive signals are provided at the knee by means of a brace.

### *1. Gait-related risk factors*

Although numerous studies have been addressing the role of an aberrant foot posture and movement and subsequent lower leg kinematics in the development of PFP, it is clear from the literature that there is still a lot of controversy concerning this issue.

The first aim of this doctoral project was therefore to gain a better insight into gait-related intrinsic risk factors which may predispose an individual to the development of the patellofemoral dysfunction syndrome.

A possible reason for the lack of consensus in the literature concerning the role of an abnormal foot posture and motion as well as other potential risk factors in the development of PFP is that the greater part of the studies which have been investigating these risk factors have been collecting information in a retrospective way. However, because of their retrospective design, it is unclear whether the deficits found in these studies are a cause or a consequence of the injury. Consequently, in the current literature a clear consensus concerning the role of many potential risk factors in the development of PFP has not been reached and many contradictions can be found between studies.

The prospective cohort study design is a more preferable study design to investigate risk factors for a specific type of injury, because only longitudinal prospective studies can determine causative relationships.(16) These kinds of studies involve measuring potential risk factors before injuries occur, after which new cases of injuries are reported during a period of follow up.(1) However, today, studies which have been investigating potential risk factors for the development of the patellofemoral dysfunction syndrome in a prospective way are rather scarce.

It has been assumed that biomechanical abnormalities in gait play an important role in the aetiology of PFP. However, despite the believe that a disruption of accurate positioning of the foot during walking and running plays a role in the development of PFP, only very few prospective studies have been undertaken to determine the role of gait related risk factors in the development of the patellofemoral dysfunction syndrome.

Two populations, in which high incidences of PFP are reported, are military recruits and runners. (15,53) Since extensive marching represents the majority of the activities military recruits perform, we were interested in investigating whether certain aberrations in the roll-over pattern of the foot during walking may be predisposing for the development of PFP.

Novice recreational runners form another group which is a considerable population at risk for PFP, due to the increasing interest of the general public in running. Hence, since running is a sport in which PFP is commonly reported, we were interested to investigate which patterns of foot roll-over could be predisposing for PFP during running, when the foot is exposed the higher impact forces compared to walking.

Therefore, in **chapters 2 and 3** of this dissertation, two prospective studies were set up to obtain a better insight in possible mechanical intrinsic gait-related risk factors for the development of PFP during walking and running, respectively. In **chapter 2**, a prospective study was set up in military recruits to investigate intrinsic risk factors inherent to the rollover pattern of the foot during gait. The gait pattern of eighty-four officer cadets was examined before the start of a six week basic military training period in which the recruits followed the same training program, mainly consisting of marching with backpacks, military tactical exercises and drills.

The most striking findings of this investigation were that during gait a more laterally directed pressure distribution underneath the foot at initial foot contact and a slower shift of the centre of pressure (COP) from the lateral side to the medial side of the foot during foot rollover were identified as predisposing factors for the development of PFP. These results suggest that the individuals who developed PFP had a more lateral oriented heel strike and rolled over their feet more on the lateral side of the foot than did the persons who remained free from PFP. We hypothesised that two mechanisms could be responsible for the onset of PFP as a consequence of the laterally oriented rollover pattern of the foot. First, the tendency to exert a more laterally oriented rollover pattern of the foot may result in decreased shock absorption of the foot as a consequence of a diminished foot pronation during the initial support phase. Freychat et al. reported a relationship between rearfoot and forefoot orientation, partially determined by the supinatory or pronatory position of the rearfoot, and ground reaction force parameters. They concluded that the specific spatial orientation of the rearfoot and forefoot can influence an 'open' and 'closed' foot behaviour, with a laterally rotated forefoot (open foot) being associated with an everted and more flexible foot, whereas a medially rotated forefoot (closed foot) was associated with a inverted and rigid foot.(8) Consequently, in the

recruits who developed PFP in our study, a more rigid position of the foot during foot rollover may have lead that higher ground-reaction forces were transferred to the knee and patellofemoral joint, placing an excessive load on the joint. This overload of the patellofemoral joint may cause a decrease of the “envelope of function” of the joint, resulting in the loss of tissue homeostasis and a subsequent onset of PFP.

Secondly, decreased pronation of the foot during the initial stance phase of gait may cause a biomechanical coupled diminished internal rotation of the tibia, which, theoretically, would place the tibial tuberosity more laterally relative to the femur resulting in an increase of the dynamic Q-angle and, hence, a larger lateral force vector on the patella.

A similar mechanism as the first gait related mechanism suggested for the onset of PFP during walking in **chapter 2** could also be concluded from the results for the running analysis in **chapter 3**. In this chapter we focussed our prospective investigation on gait related intrinsic risk factors for PFP by evaluating static foot posture and measuring plantar pressure during running in 102 recreational runners. The results of this study revealed that an excessive vertical peak force underneath the lateral heel and underneath the second and third metatarsal during running were discriminating factors between the 17 runners who sustained PFP and those who did not. In accordance with the first proposed aetiological mechanism for the development of PFP in the military recruits in **chapter 2**, in **chapter 3** an excessive impact shock could be assumed as a causal factor for the onset of PFP in the injured runners. The excessive vertical peak forces underneath the lateral heel during heel strike and underneath the second and third metatarsal during the propulsion phase of running presumably cause that higher impact shocks are transferred to the patellofemoral joint. As assumed in **chapter 2**, this may lower the threshold for the initiation of the loss of tissue homeostasis in this joint.

The more laterally directed pressure distribution underneath the foot at initial foot contact and slower shift of the centre of pressure from the lateral side to the medial side of the foot, which were identified as risk factors for the development of patellofemoral pain during walking in the military recruits (chapter 2) were however not found in the running-analysis of the investigated runners in chapter 3. In other words, during running the runners who developed patellofemoral pain in chapter 3 did not show the laterally oriented rollover pattern of the foot suggesting insufficient foot pronation, which we previously identified as being a risk factor for the development of patellofemoral pain during walking. Hence, based upon the results of the studies in chapters 2 and 3 one might conclude that the rollover pattern of the foot which predisposed persons to the development of PFP during walking does not equal the rollover

pattern of the foot which seemed to be contributing to developing PFP during running. However, in a retrospective study, Duffey et al. (49) found that also runners, afflicted with PFP, pronated less through the initial stance phase of running, which does agree with our results of the gait-analysis of the recruits who developed PFP during walking (chapter 2).

Several factors may however account for the differences in results, which were found between chapters 2 and 3 of this dissertation. The fact that the laterally oriented rollover pattern of the foot found during walking was not found during running may be the result of the occurrence of possible functional differences in foot unroll between running and walking. Although De Cock et al. (51) found that the distribution of the pressure over the foot during running is comparable to that of walking and consequently concluded that in both locomotion forms, the functional foot behaviour is almost identical in walking and running, Rosenbaum et al. (52) showed that total foot loading shifts medially and hind foot eversion increases as walking speed increases.

In addition, the recruits in chapter 2 were all physically active, predominantly male subjects, whereas the subjects in chapter 3 were predominantly untrained novice female runners. Possibly the differences in gender and exercise status between both groups also may have had an influence on the differences in study results due to possible constitutional differences (e.g. influence of a wider pelvis in women on frontal plane knee angle and foot posture) and possible differences in movement patterns. Cho et al. demonstrated that differences in anatomical structure between men and women cause gender differences in hip, knee and ankle kinematics during the stance phase of walking and running.(50)

A remarkable finding in chapter 2 was that in this population of military recruits 58% of the female recruits developed PFP versus 38% of the male recruits. This finding is in accordance with the literature which indicates that women are more commonly afflicted with this problem than are men. (54,55,56,57) However, although this was not reported in chapter 2, no significant differences were found when the plantar pressure data of the male and female recruits were compared. Possibly, other structural, muscular or sociologic differences between the male and female recruits, may have had an influence on the difference in incidence of PFP between both genders in this study. Indeed, the higher incidence of PFP in women has been attributed to gender differences in anatomic structure, muscle strength and sociologic factors. (54,55) The most obvious reason why women would tend to be more afflicted with PFP is the difference in lower extremity alignment. (54) The pelvis of a female is larger relative to the individual's overall structure. The broader pelvis moves the hip joints farther lateral relative to the midline and therefore produces an increased valgus angle from the hip to the knee and

then to the ground. In addition to a broader pelvis, females have a higher prevalence of increased femoral anteversion. (58) This increase in femoral anteversion and a wider pelvis also are associated with an increased Q-angle. (59) Because the lower extremity muscles (most notably the quadriceps) follow the orientation of the femur, the knee extensor mechanism in women generally has a greater valgus orientation than in men. This valgus load onto the patella causes an increase of the pressure on the lateral facet of the patella. This increased valgus thrust on the patella may increase the tendency of excessive lateral pressure on the patella, which may increase the risk of developing PFP. Also, possible neuromuscular differences between the male and female recruits may have accounted for the gender discrepancy in PFP incidence in the study of chapter 2. Neuromuscular differences in the lower extremity have been demonstrated between men and women. (60) Women have been indicated to demonstrate less muscle mass and different muscle fibre compositions. (61,62) It is also known that hormonal influences, particularly testosterone, can increase muscle mass, fiber recruitment and a proportion of type II fibers, which favours muscle strength increases in men over women. (63) Furthermore, differences in sociologic factors between the male and female recruits may also have accounted for the remarkable difference in incidences. Lower tolerance of pain has been implicated as a factor influencing the increased incidence of PFP in women.(54) Also, McAlindon et al. reported that women are more likely to report a disability.(64)

However, these factors, which may have had an influence on the difference in incidence of PFP between the male and female recruits, were not assessed in our study of chapter 2.

Our findings in **chapters 2 and 3** are not in accordance with the conclusions of other studies which have associated the incidence of PFP with the quantity or timing of subtalar pronation.(6,13,21,27,41) Although authors have suggested that excessive (6,21,27,41) or prolonged (13) foot pronation causes PFP, this was not supported by the findings in our studies. In contrast, a more laterally oriented rollover pattern of the foot during walking was identified as a risk factor for the development of PFP in military recruits (**chapter 2**). Furthermore, the results of both studies in **chapters 2 and 3** rather suggest that an increased loading of the patellofemoral joint as a result of a decreased shock absorbing capacity of the foot or an increased impact shock at the early contact phase and during the propulsion phase of the foot during running plays an important role in the aetiology of PFP, as pointed out above. A possible explanation for the difference in results between our study in chapter 2 and previous studies may however be that the military recruits in our study wore rigid combat shoes during the military training activities such as marching, which could have had an

influence on the roll-over pattern of the recruits feet. Indeed, it could have been possible that in recruits who would normally tend to exert an excessive pronation of the foot during walking, this roll-over pattern of the foot was limited during activities by the rigid nature of the soles and other parts of the used combat shoes, which were used during the military training activities. In addition, the rigid nature of the used combat shoes during training, with poor shock absorbing capacities, may have enforced the effect of the decreased shock absorbing capacity of the feet of the recruits who showed an excessive laterally oriented roll-over pattern of the foot during walking and consequently may have additionally accounted for an increased impact shock, leading to an overload at the knee in the subjects who developed PFP.

When comparing the results of the studies in **chapters 2 and 3** with previous studies in the literature, the remark should however be made that, together with a study by Hetsroni et al (15) our studies, at present are the only studies which have investigated the relationship between the rollover pattern of the foot and PFP in a prospective manner.

Besides the assumed association between foot motion during gait and the incidence of PFP, several authors have reported on variances in static foot alignment as a possible contributing factor for the development of PFP. (25,35) A perusal of the literature shows however that the relationship between the static posture of the foot and PFP remains controversial. (25,31,35,47) Therefore, in **chapter 3** of this dissertation we were interested in investigating in a prospective way whether variances in the static standing foot posture of runners are correlated with the incidence of PFP. Out of the results of this study the conclusion could be drawn that there was no significant evidence to support the assumption that an excessively pronated or supinated foot posture in standing position is associated with the development of PFP. This result is in line with another prospective study by Witvrouw et al., which concluded that static foot posture is not a predictive factor for the development of PFP.(47)

When taking the static alignment of the foot into consideration as a possible aetiological factor for the onset of PFP, the question arises however what predictive value static foot alignment measurements have for dynamic situations. It has been assumed that a given structural foot type will display certain functional characteristics and these, in turn, will be related to pathomechanics of the foot and the lower extremity.(37) Consequently, attempts have been made to predict dynamic foot function by using static measurements. Levinger and Gilleard stated that clinical rearfoot measurement of relaxed standing can be used to explain the pattern of rearfoot motion during walking. (26) However, some researchers have seriously

questioned the validity of static measurements to predict dynamic foot functional behaviour. (14,17,22,29) McPoil and Cornwall investigated the relationship between static lower extremity measurements and rearfoot motion during walking and concluded that static measurements of the foot are poor predictors of dynamic rearfoot motion as measured by maximum pronation or time to maximum pronation.(29) This conclusion is supported by Hunt et al. who also reported that static measurements of calcaneal deviation and medial arch angle are limited in their ability to predict three-dimensional rearfoot movement during walking.(17) Consequently, nevertheless some retrospective studies have reported on a possible association between static foot alignment and the occurrence of PFP (25,35), given the above reported data, it is still questionable whether static measurements of foot posture are an efficient modality to predict abnormal foot kinematics during gait.

Although this was not reported in chapter 3, in the studied population of this chapter no significant correlations ( $p < 0.05$ ) were found between the subjects' standing foot posture and the foot roll-over pattern during running. The highest correlations found were between the foot posture index (FPI) data and the maximal force underneath the lateral heel ( $r = -0.217$ ) and between the FPI data and the time to the maximal force underneath the first metatarsal ( $r = -0.266$ ). However, with such low correlation coefficients, it can be stated that in the investigated population of the study in chapter 3 no meaningful correlations were present between the subjects' standing foot posture and their foot roll-over pattern during running.

Hence, the fact that static foot posture measurements may not be good predictors for an aberrant dynamic foot function may explain why no correlation was found between the static standing foot posture and the development of PFP in our study of chapter 3. Therefore, it is our opinion that care must be taken in attributing patellofemoral problems to observed deviations in static foot posture. However, since relatively little is yet known about the effect of foot type on the plantar pressure distribution pattern, the fact whether or not different foot types reflect in clinically important changes of plantar pressure pattern during (running) gait needs to be more extensively investigated.

Based on the findings in **chapters 2 and 3** of this dissertation, we recommend that in order to decrease the risk for developing PFP, the gait related risk factors, identified in this doctoral project should be taken into account as possible contributing factors in the onset of this disorder. Therefore, we believe it would be advisable that the assessment of the rollover pattern of the foot during walking and running would be integrated in the screening process of athletic and active populations at risk for PFP, such as runners and military personnel, in



addition to other described clinical evaluations which have been advised in the literature for the detection of risk factors for this pathology.(9,23,46,48)

In subjects who are susceptible for PFP, we suggest that an excessive laterally oriented rollover pattern of the foot and excessive vertical peak force during heel strike and forefoot push off should be adjusted. The use of orthotics is frequently prescribed in the belief that they correct the biomechanical dysfunction of specific joints of the lower extremity. Functional orthoses are usually prescribed in an attempt to alter foot function with the expectation that they will guide the foot through the weight-bearing stance phase of gait to promote overall biomechanical efficiency. Although the mechanics by which orthoses are sometimes effective are not fully understood, a significant reduction in pain with the use of orthoses has been reported in PFP patients.(6,39,40) Medially posted, custom-made soft orthoses have been shown to change transverse and frontal plane movements of the foot and ankle during treadmill walking and running in a group of patients with PFP.(7) Eng & Pierrynowski reported that in patients suffering from PFP, the use of orthotics, correcting excessive pronation of the foot, in addition to an exercise programme was more effective than an exercise programme alone.(6) Leung et al. indicated that the effectiveness of orthotics lies in aligning the orientation and movements of the subtalar, ankle and knee joints by reducing the degree and duration of abnormal pronation.(24) Consequently, it has been suggested in the literature that patients with PFP may benefit from foot orthoses if they also demonstrate signs of excessive foot pronation.(11) We believe however that the same line of reasoning may also be true for PFP patients or individuals at risk for PFP, who demonstrate the intrinsic gait related risk factors identified in our studies. It is our opinion that presumably both deviations of the normal rollover pattern of the foot, excessive pronation as well as insufficient pronation, may cause patellofemoral dysfunction as both mechanisms may lead to overload of the PF-joint. However, the presumed effect of orthotics, correcting an excessive laterally oriented rollover pattern of the foot and excessive vertical peak forces during heel strike and forefoot push off, on PFP needs further scrutiny.

## *2. Correlation between hip-muscle strength and frontal plane knee-motion*

The second objective of this doctoral project was to gain a better insight in the relationship between hip muscle strength and the frontal plane movement of the knee.

Weakness of the hip abductor, external rotator, and hip flexor muscles has been suggested to be a possible causal factor for the development of PFP.(18,38,42) It is theorized that weakness of these muscles may increase femoral internal rotation, adduction, and valgus knee movements. These deviations may lead to an increased Q-angle, which may subsequently

alter the tracking of the patella, increase compressive forces on the patellofemoral joint, and ultimately lead to PFP.(36,42) Although hip muscle weakness has been hypothesised to result in an increased Q-angle through increased hip internal rotation, adduction, and knee valgus, to our knowledge, no studies have investigated the relationship between hip muscle strength and knee kinematics during dynamic tasks. Consequently, our study (**chapter 4**) was the first to examine whether the strength of the muscles around the hip joint is related to the frontal plane motion of the knee during a dynamic task. The strength of the hip flexor, extensor, abductor, adductor, internal rotator, and external rotator muscles was measured in 84 healthy subjects, using a handheld dynamometer. Subsequently, the subjects were videotaped during a forward lunge movement and peak knee valgus and varus angles were determined by means of a digital video analysis software program.

The results of the study (**chapter 4**) showed no significant correlation between the muscle strength of any of the six tested muscle groups and the amount of knee valgus or varus during the functional lunge movement. Although previous studies have speculated on the relationship between hip muscle strength and altered knee kinematics, this assumption could not be confirmed by the results in **chapter 4**. Prior to our study, only one study by Mascal et al. examined the interrelationship between hip strength and hip kinematics in one single subject.(28) As stated earlier in the general introduction of this dissertation, this study provided preliminary evidence to support the relationship between hip muscle weakness and lower extremity kinematics (e.g. excessive hip internal rotation and adduction), but lacked information regarding the correlation between hip muscle strength and knee kinematics. The findings of our study however suggest that variations in hip muscle strength may not necessarily result in altered knee kinematics. The results of our study are supported by a recent study by Bolgla et al., who examined the relationship between hip muscle strength and knee frontal plane angles during stair descent in PFP patients.(4) These authors reported that the examined patients with PFP demonstrated significant hip weakness but did not demonstrate altered knee kinematics.

Consequently, out of the results of our study and the study by Bolgla et al. (4) it can be concluded that hip muscle strength may not influence knee kinematics as hypothesised in previous retrospective studies which have been investigating the relationship between hip muscle weakness and PFP.

### *3. Influence of knee bracing on brain activity during knee movement*

Knee braces are frequently used in the treatment of PFP. Although the application of a brace appears to be effective in reducing patellofemoral pain, the underlying mechanism is not

entirely clear. Besides its assumed mechanical function on patellar tracking, the beneficial effect of knee bracing is believed to be related to their enhancement of joint proprioception.(3,10,12,33,34,44) The application of a knee brace is assumed to stimulate several receptors (Ruffini receptors, Pacinian corpuscles, Golgi tendon organs, free nerve endings, muscle spindles) in the knee joint capsule and ligaments, muscles and tendons surrounding the knee, and skin. Their afferent inputs are believed to be integrated at all levels of the central nervous system to generate appropriate motor responses. Consequently, it has been suggested in the literature that enhanced proprioceptive input to the knee may contribute to the recruitment of the muscles surrounding the joint (43) which may, in turn, enhance patellar tracking. However, it is still obscure at which level of the central nervous system this mechanism is regulated. Although research has been conducted on the central processing of proprioceptive signals during passive and active upper limb movement (5,32,45), to our knowledge, no studies have investigated which area of the brain is involved in the central processing of incoming proprioceptive information during movement of the knee.

In **chapter 5** of this dissertation we investigated if varying proprioceptive inputs to the knee by means of a moderately tight knee sleeve and tight knee brace affect the degree to which the higher motor control centres of the brain are activated. Out of the results of this study it could be concluded that the primary sensorimotor cortex is involved in the central processing of proprioceptive information coming from the knee. The significantly higher activation of this cortical area during knee movement while wearing a knee brace compared to the degree of activation when a sleeve or no brace were applied reveals that proprioceptive information coming from the knee joint is conveyed to the primary sensorimotor cortex and that the degree to which this cortical area is activated can be influenced by varying proprioceptive stimulation at the knee. Furthermore, the activated area of the brain during knee movement without application of a brace or sleeve corresponded well to the brain area which showed an increased activation when a brace and sleeve, respectively, were applied. This finding may indicate that peripheral somatosensory stimulation by means of a knee brace or sleeve stimulates the motor areas which are responsible for the generation of corresponding limb movement and, hence, controlling the muscles around the implicated joint. As it has been suggested that cutaneous stimulation by means of taping or bracing seems to have an impact on muscle recruitment, the findings of this study may offer a plausible explanation for the mechanism by which bracing may enhance the neuromuscular control of the patellofemoral joint.

## **Clinical relevance of this doctoral project.**

As postulated in the beginning of this dissertation, alignment problems within and outside the patellofemoral joint in the lower extremity are considered to be the most important aetiological mechanisms for the development of PFP. Concerning alignment problems outside the PF-joint, dysfunctions leading to malalignment in the segments distal and proximal to the PF-joint have been propounded in the literature to have an influence on the biomechanics of the PF-joint and, hence, on the development of patellofemoral pain. (6,13,18,21,27,38,41,42) However, due to a lack of prospective studies in this regard, no consensus has been reached concerning the relationship between dysfunctions and altered biomechanics at the hip joint and foot and the onset of patellofemoral problems. In addition, improving the proprioceptive ability of the knee joint is another factor which is thought to have an important influence on PFP because of its assumed contribution to the neuromuscular control of patellar tracking. However, in the present literature information is still lacking concerning the mechanism by which this process is regulated.

The aims in the present dissertation were therefore threefold: 1) to prospectively investigate whether certain variables, inherent to the roll-over pattern of the foot, might be predisposing for the development of PFP during walking and during running 2) to assess whether hip muscle strength is related to the amount of frontal plane knee movement, affecting patellofemoral biomechanics, during a functional lower limb movement and 3) to investigate to which degree brain activity is influenced during knee movement when additional proprioceptive input is applied at the knee by means of the application of a brace.

The results of this doctoral project contribute to a) an extension of the knowledge concerning the roll of intrinsic variables affecting lower extremity alignment in the aetiology of PFP and b) may offer a possible explanation for the mechanism by which enhanced proprioceptive input to the knee by means of a brace might enhance the neuromuscular control of the patellofemoral joint. The results of our investigations should be considered as valuable information, which might correlate with other clinical findings in the examination of the PFP-patient in order to understand the malalignment problem in the aetiology of PFP as fully as possible.

## **Strengths and limitations of the studies and future directions.**

The aim of this doctoral project was to obtain a better insight into the intrinsic risk factors for the development of PFP. In this project we focussed on certain dysfunctions in the joints lying distal and proximal to the patellofemoral joint. It was also our purpose to explore how improvement of knee joint proprioception by means of a brace, which is presumed to be an important modality in the conservative treatment of PFP, might be regulated.

**Chapter 2** is the first study to prospectively examine potential gait-related risk factors for the development of the patellofemoral dysfunction syndrome. Former studies have assessed potential causal gait-related factors for PFP in a retrospective way. However, as pointed out earlier in the general discussion, to investigate cause-and-effect relationships, it is necessary that potential risk factors for an injury are examined in subjects before the occurrence of injury. Another strength of this study is the fact that the study was conducted on military recruits, following the same military training in the same conditions, with the same equipment. This ensured us that the influence of extrinsic factors was kept under control as much as possible.

A limitation of this study, and the study in **chapter 3**, was however that not all contributing factors were measured. It should be kept in mind that the aetiology of PFP is multifactorial and other possible intrinsic risk factors probably also could have influenced the onset of the pathology. The selection of the investigated potential risk factors was however based on a perusal of retrospective studies which have been suggesting associations between these variables and PFP. The procedures used to measure these variables were selected according to the reliability and availability of the equipment.

The high incidence of PFP found in **Chapter 2**, could be attributed to the physically heavy training regime the recruits underwent during the basic military training period. The fact that a specific study population was examined may form a limitation of the study because care must be taken when extrapolating the results of our study to a general population with a different exercise status. On the other hand, this clearly defined study sample of military recruits was chosen in order to make it possible to control the many possible extrinsic risk factors. In **chapter 3**, gait-related risk factors for PFP during running were investigated in a study population consisting of recreational novice runners. This group formed a more representative study sample for the general population. However, this involved that certain possible risk factors, such as running equipment, could be less controlled for in this study. Nevertheless, out of the investigations in **chapters 2 and 3** various potential gait-related risk factors for the

development of PFP have been established. In future research, prospective studies of intervention programs, which focus on correcting the intrinsic gait-related risk factors identified in **chapters 2 and 3**, are needed to investigate whether an alteration of these factors proves to be effective in the prevention and treatment of PFP.

**Chapter 4** is the first study which examined the interrelationship between hip muscle strength and the movement of the knee in the frontal plane. In this study the isometric strength of the hip muscles was tested in asymptomatic healthy individuals. It might be possible that in this population no correlation between hip muscle strength and knee kinematics was found because these healthy subjects without lower limb complaints had sufficient hip strength to maintain the frontal plane knee alignment during the forward lunge movement. It is plausible that a correlation between the investigated variables could manifest itself in symptomatic subjects presenting severe hip muscle weakness. It remains however elusive in retrospective studies, which have been investigating the relationship between hip muscle weakness and PFP, if this weakness of the hip muscles and presumed altered hip and knee kinematics was already present prior to developing PFP. Therefore, the primary goal of our study was to search for a possible relationship between hip muscle strength and the frontal plane movement of the knee during a functional movement in an asymptomatic population. Recently, a study by Bolgia et al. investigated hip strength and hip and knee kinematics during stair descent in persons with PFP presenting hip muscle weakness but also did not demonstrate altered knee kinematics in these subjects.<sup>(4)</sup> However, since it remains unclear if hip muscle weakness might be a cause or a consequence of PFP, future prospective studies are necessary for addressing this question. Another possible reason for our finding in **chapter 4** may be related to the chosen task. We examined frontal plane knee movement while the subjects exerted a forward lunge movement. This task may not have been challenging enough, i.e. the subjects having sufficient hip strength to maintain the frontal plane knee alignment during this manoeuvre. Subjects might have exhibited altered lower extremity mechanics if assessed during a more challenging task. In addition, it might be possible that the investigated relationship only reveals itself when the muscles are getting fatigued. Therefore, future research should assess whether this relationship might be present when the hip muscles are fatigued during prolonged exercise or when they are tested during more challenging manoeuvres.

In **chapter 5**, we were the first to explore whether and to which degree the higher motor control centres in the brain are influenced by additional proprioceptive signals coming from the knee by means of brace application. In this chapter we hypothesised that by stimulating

the primary sensorimotor cortex, additional proprioceptive input to the knee caused by the application of a knee brace may contribute to controlling the muscles around the knee and enhance the neuromuscular control of this joint. Future studies should assess whether PFP-patients showing a deficit in knee proprioception demonstrate differences in primary sensorimotor cortex activity during knee movement in comparison with asymptomatic individuals. Once this is known, it would be interesting to investigate whether a possible difference in cortical activation in these patients correlates with an alteration in lower extremity alignment. This might contribute significantly to the way of thinking about the role of motor control in the pathogenesis of patellofemoral pain syndrome.

A limitation of the study was that the subjects performed an open chain exercise whereas a closed chain movement would have been more functional for the lower extremity. However, due to the fact that functional magnetic resonance imaging (fMRI) was used in order to investigate differences in brain activity, it was impossible to perform this study in a weight bearing position as the subjects needed to be positioned supine into the MRI-scanner during imaging. In addition, in order to collect usable MRI data, head motion needed to be restricted within 4 millimeters. Using a bit-bar devise and additional straps around the forehead and hips we were able to restrain the subjects' head within the 4 millimeter limit during the open chain knee movement. However, this would not have been achieved in a closed chain situation.

## REFERENCES

1. Bahr R, Holme I. Risk factors for sports injuries – a methodological approach. *Br J Sports Med.* 2003; 37: 384-392.
2. Baker V, Bennel K, Stillman B, Cowan S, Crossley K. Abnormal knee joint position sense in individuals with patellofemoral pain syndrome. *J Orthop Res.* 2002; 20(2): 208-214.
3. Birmingham TB, Inglis JT, Kramer JF, Vandervoort AA. Effect of a neoprene sleeve on knee joint kinesthesia: comparison of active, passive and axially loaded joint angle replication tests. *Med Sci Sports Exerc.* 2000; 32: 304-308.
4. Bolgla AL, Malone TR, Umberger BR, Uhl TL. Hip strength and hip and knee kinematics during stair descent in females with and without patellofemoral pain syndrome. *J Orthop Sports Phys Ther.* 2008; 38(1): 12-18.
5. Dettmers C, Fink GR, Lemon RN, Stephan KM, Passingham RE, Silbersweig D, Holmes A, Ridding MC, Brooks DJ, Frackowiak RS. Relation between cerebral activity and force in the motor areas of the human brain. *J Neurophysiol.* 1995; 74: 802-815.
6. Eng JJ, Pierrynowski MR. Evaluation of soft foot orthotics in the treatment of patellofemoral pain syndrome. *Phys Ther.* 1993; 73(5): 62-68.
7. Eng JJ, Pierrynowski MR. The effects of soft foot orthotics on three-dimensional lower limb kinematics during walking and running. *Phys Ther.* 1994; 74(9): 836-844.
8. Freychat P, Belli A, Carret JP, Lacour JR. Relationship between rearfoot and forefoot orientation and ground reaction forces during running. *Med Sci Sports Exerc.* 1996; 28(2): 225-32.
9. Fulkerson JP. Diagnosis and treatment of patients with patellofemoral pain. *Am J Sports Med.* 2002; 30(3): 447-456.
10. Gilleard W, McConnell J, Parsons D. The effect of patellar taping on the onset of vastus medialis obliquus and vastus lateralis muscle activity in persons with patellofemoral pain. *Phys Ther.* 1998; 78: 25-32.
11. Gross MT, Foxworth JL. The role of foot orthoses as an intervention for patellofemoral pain. *J Orthop Sports Phys Ther.* 2003; 33(11): 661-670.
12. Guling LK, Lephart SM, Stone DA, Irrgang JJ, Pincivero DM. The effect of patellar bracing on quadriceps EMG activity during isokinetic exercise. *Isokin Exerc Sci.* 1996; 6: 133-138.



13. Hamill J, Bates BT, Holt KG. Timing of lower extremity joint actions during treadmill running. *Med Sci Sports Exerc.* 1992; 24(7): 807-13.
14. Hamill J, Bates BT, Knutzman KM. Relationship between selected static and dynamic lower extremity measures. *Clin Biomech.* 1989; 4: 217-25.
15. Hetsroni I, Finestone A, Milgrom C, Ben Sira D, Nyska M, Radeva-Petrova D, Ayalon M. A prospective biomechanical study of the association between foot pronation and the incidence of anterior knee pain among military recruits. *J Bone Joint Surg.* 2006; 88(7): 905-908.
16. Hodgson Phillips L. Sports injury incidence. *Br J Sports Med.* 2000; 34: 133-136.
17. Hunt AE, Fahey AJ, Smith RM. Static measures of calcaneal deviation and arch angle as predictors of rearfoot motion during walking. *Australian Journal of Physiotherapy.* 2000; 46(1): 9-16.
18. Ireland ML, Willson JD, Ballantyne BT, Davis IM. Hip strength in females with and without patellofemoral pain. *J Orthop Sports Phys Ther* 2003; 33(11): 671-76.
19. Jerosch J, Schmidt K, Prymka M. Proprioceptive capacities of patients with retropatellar knee pain with special reference to effectiveness of an elastic knee bandage. *Unfallchirurg.* 1997; 100(9): 719-723.
20. Keenan AM, Redmond AC, Horton M, Conaghan PG, Tennant A. The Foot Posture Index: Rasch analysis of a novel footspecific outcome measure. *Arch Phys Med Rehabil.* 2007; 88: 88-93.
21. Klingman RE, Liaos SM, Hardin KM. The effect of subtalar joint posting on patellar glide position in subjects with excessive rearfoot pronation. *J Orthop Sports Phys Ther.* 1997; 25(3): 185-191.
22. Knutzen KM, Price A. Lower extremity static and dynamic relationship with rearfoot motion in gait. *J Am Podiatr Med Assoc.* 1994; 84(4): 171-80.
23. LaBella C. Patellofemoral pain syndrome: evaluation and treatment. *Prim Care Clin Office Pract.* 2004; 31: 977-1003.
24. Leung AK, Mak AF, Evans JH. Biomechanical gait evaluation of the immediate effect of orthotic treatment for flexible flat foot. *Prosthet Orthot Int.* 1998; 22(1): 25-34.
25. Levinger P, Gillear W. An evaluation of the rearfoot posture in individuals with patellofemoral pain syndrome. *J Sports Sci and Med.* 2004; 3: 8-14.
26. Levinger P, Gillear W. Relationship between static posture and rearfoot motion during walking in patellofemoral pain syndrome-effect of a reference posture for gait analysis. *Journal of the American Podiatric Medical Association.* 2006; 96(4): 323-329.

27. Lun V, Meeuwisse W, Stergiou P, Stefanyshyn D. Relation between running injury and static lower limb alignment in recreational runners. *Br J Sports Med.* 2004; 38: 576-580.
28. Mascal CL, Landel R, Powers C. Management of patellofemoral pain targeting hip, pelvis and trunk muscle function: 2 case reports. *J Orthop Sports Phys Ther* 2003; 33(11): 647-60.
29. McPoil TG, Cornwall MW. The relationship between static lower extremity measurements and rearfoot motion during walking. *J Orthop Sports Phys Ther.* 1996; 24(5): 309-314.
30. Mellor R, Hodges PW, 2006. Effect of knee joint angle on motor unit synchronization. *J Orthop Res.* 24, 1420-1426.
31. Messier SP, Davis SE, Curl WW, Lowerly RB, Pack RJ. Etiologic factors associated with patellofemoral pain in runners. *Med Sci Sports Exerc.* 1991; 23 (9): 1008-1015.
32. Mima T, Sadato N, Yazawa S, Hanakawa T, Fukuyama H, Yonekura Y, Shibasaki H. Brain structures related to active and passive finger movements in man. *Brain.* 1999; 122: 1989-1997.
33. Osternig LR, Robertson RN. Effects of prophylactic knee bracing on lower extremity position and muscle activation during running. *Am J Sports Med.* 1993; 21: 733-737.
34. Perla R, Frank C, Fick G. The effect of elastic bandages on human knee proprioception in the uninjured population. *Am J Sports Med.* 1995; 23: 251-255.
35. Powers CM, Maffucci R, Hampton S. Rearfoot posture in subjects with patellofemoral pain. *J Orthop Sports Phys Ther.* 1995; 22 (4): 155-160.
36. Powers CM. The influence of altered lower-extremity kinematics on patellofemoral joint dysfunction: A theoretical Perspective. *J Orthop Sports Phys Ther.* 2003; 33: 639-646.
37. Razeghi M, Batt ME. Biomechanical analysis of the effect of orthotic shoe inserts: A review of the literature. *Sports Med.* 2000; 29(6): 425-438.
38. Robinson RL, Nee RJ. Analysis of hip strength in females seeking physical therapy treatment for unilateral patellofemoral pain syndrome. *J Orthop Sports Phys Ther.* 2007; 37(5): 232-238.
39. Saxena A, Haddad J. The effect of foot orthoses on patellofemoral pain syndrome. *J Am Podiatr Med Assoc.* 2003; 93(4): 264-271.
40. Sutlive TG, Mitchell SD, Maxfield SN, McLean CL, Neumann JC, Swiecki CR, Hall RC, Bare AC, Flynn TW. Identification of individuals with patellofemoral pain whose

- symptoms improved after a combined program of foot orthosis use and modified activity: A preliminary investigation. *Phys Ther.* 2004; 84(1): 49-61.
41. Tiberio D. The effect of excessive subtalar joint pronation on patellofemoral mechanics: A theoretical model. *J Orthop Sports Phys Ther.* 1987; 9: 160-165.
  42. Tyler TF, Nicholas SJ, Mullaney MJ, McHugh MP. The role of hip muscle function in the treatment of patellofemoral pain syndrome. *Am J Sports Med.* 2006; 35: 1-7.
  43. Van Tiggelen D, Coorevits P, Witvrouw E. The effects of a neoprene knee sleeve on subjects with a poor versus good joint position sense subjected to an isokinetic fatigue protocol. *Clin J Sport Med.* 2008; 18 (3): 259-265.
  44. Van Tiggelen D, Coorevits P, Witvrouw E. The use of a neoprene knee sleeve to compensate the deficit in knee joint position sense caused by muscle fatigue. *Scand J Med Sci Sports.* 2008; 18: 62-66.
  45. Weiller C, Juptner M, Fellows S, Rijntjes M, Leonhardt G, Kiebel S, Muller S, Diener H, Thilmann A. Brain representation of active and passive movements. *Neuroimage.* 1996; 4: 105-110.
  46. Werner S. Conservative treatment of athletes with anterior knee pain. *Science: Classical and new ideas.* In: *Anterior knee pain and patellar instability.* Vicente Sanchis-Alfonso, ed. Springer, London. 2006: 147-166.
  47. Witvrouw E, Lysens R, Bellemans J, Cambier D, Vanderstraeten G. Intrinsic risk factors for the development of anterior knee pain in an athletic population: A two-year prospective study. *Am J Sports med.* 2000; 28 (4): 480-489.
  48. Witvrouw E, Werner S, Mikkelsen C, Van Tiggelen D, Vanden Berghe L, Cerulli G. Clinical classification of patellofemoral pain syndrome: guidelines for non-operative treatment. *Knee Surg Sports Traumatol Arthrosc.* 2005; 13: 122-130.
  49. Duffey MJ, Martin DF, Cannon DW, Craven T, Messier SP. Etiologic factors associated with anterior knee pain in distance runners. *Med Sci Sports Exerc.* 2000; 32 (11): 1825-1832.
  50. Cho Sh, Park JM, Kwon OY. Gender differences in three-dimensional gait analysis data from 98 healthy Korean adults. *Clin Biomech.* 2004; 19: 145-152.
  51. De Cock A, Willems T, Witvrouw E, Vanrenterghem J, De Clercq D. A functional foot type classification with cluster analysis based on plantar pressure distribution during jogging. *Gait and Posture.* 2006; 23: 339-347.
  52. Rosenbaum D, Hautmann S, Gold M, Claes L. Effects of walking speed on plantar pressure patterns and hindfoot angular motion. *Gait and Posture.* 1994; 2: 191-197.

53. Cheung RTH, Ng GYF, Chen BFC. Association of footwear with patellofemoral pain syndrome in runners. *Sports Med.* 2006; 36(3): 199-205.
54. Fulkerson JP, Arendt A. Anterior knee pain in females. *Clin Orthop.* 2000; 372: 69-73.
55. Beim GM. Sports Injuries in women. How to minimize the increased risk of certain conditions. *Womens Health.* 1999; 2: 27-34.
56. Dehaven KE, Lintner DM: Athletic injuries: Comparison by age, sport and gender. *Am J Sports Med.* 1986; 14: 218-224.
57. Thomée R, Renstrom P, Karlsson J, Grimby G. Patellofemoral pain syndrome in young women. *Scand J Med Sci Sports.* 1995; 5: 237-244.
58. Staheli LT. Rotational problems in children. *J Bone joint Surg.* 1993; 75A: 939-949.
59. Hvid I, Andersen LI. The quadriceps angle and its relation to femoral torsion. *Acta Orthop Scand.* 1982; 53: 577-579.
60. Huston LJ, Wojtys EM. Neuromuscular differences between male and female athletes contributing to anterior cruciate ligament injuries. In: Garrett WE, Lester GE, McGowan J, et al, editors. *Women's health in sports and exercise.* Rosemont: American Academy of Orthopaedic Surgeons. 2001: 347-356.
61. Kanehisa H, Okuyama H, Ikegawa S. Sex differences in force generation capacity during repeated maximal knee extensions. *Eur J Appl Physiol.* 1996; 73: 557-562.
62. Miller AE, MacDougall JD, Tarnopolsky MA. Gender differences in strength and muscle fiber characteristics. *Eur J Appl Physiol.* 1993; 66: 254-262.
63. Ramos E, Frontera WR, Llopart. Muscle strength and hormonal levels in adolescents: gender related differences. *Int J Sports Med.* 1998; 19: 526-531.
64. Mc Alindon T, Cooper C, Kirwan J, Dieppe P. Knee pain and disability in the community. *Br J Rheumatol.* 1992; 31: 189-192.

**CHAPTER 7**  
**NEDERLANDSTALIGE SAMENVATTING**



## NEDERLANDSTALIGE SAMENVATTING

Het patellofemorale disfunctiesyndroom (PFDS) is één van de meest voorkomende aandoeningen ter hoogte van de knie. In de literatuur beschrijft men een incidentie van één op vier in de algemene populatie met een nog hogere incidentie bij sportbeoefenaars. De hoge incidentie van patellofemorale pijn (PFP) toont aan dat deze pathologie geïdentificeerd kan worden als één van de belangrijkste oorzaken van problemen en pijn ter hoogte van de knie bij fysiek actieve personen. Als gevolg van een toenemende publieke belangstelling voor sportbeoefening gedurende de laatste decennia, wordt hiermee ook een proportionele toename waargenomen van het aantal personen dat het patellofemorale disfunctiesyndroom ontwikkelt. PFP wordt algemeen beschouwd als een multifactorieel probleem dat geassocieerd wordt met mogelijke oorzakelijke factoren die solitair of in combinatie met elkaar kunnen lijden tot het ontstaan van patellofemorale klachten. Deze factoren kunnen worden onderverdeeld in extrinsieke risicofactoren (omgevingsgebonden) en intrinsieke risicofactoren (persoonsgebonden).

In het verleden werd al veel onderzoek verricht naar de mogelijke rol van disfuncties van structuren die nauw verbonden zijn met het patellofemorale gewricht (PF-gewricht) in de ontwikkeling van PFP. Hierbij werd voornamelijk gefocust op musculaire en niet musculaire structuren die een rechtstreekse invloed op het alignment van PF-gewricht uitoefenen. Malalignment van het patellofemorale mechanisme kent zijn oorzaak echter niet enkel lokaal ter hoogte van het PF-gewricht, maar wordt tevens beïnvloed door segmentale interacties ter hoogte van het onderste lidmaat. Bijgevolg is er in de meer recente jaren een toenemende interesse naar de gevolgen van disfuncties ter hoogte van de heup, enkel en voet op de biomechanica van het PF-gewricht. Abnormale bewegingen van de tibia en femur in het transversale en frontale vlak, die zouden resulteren uit een afwijkende kinematica ter hoogte van de voet, enkel en heup tijdens gaan en lopen, worden verondersteld een effect te hebben op de biomechanica van het PF-gewricht en het ontwikkelen van PFP. Tot op heden zijn echter weinig studies voorhanden die de invloed van deze potentiële risicofactoren op het ontstaan van PFP op een prospectieve manier onderzochten. Bijgevolg is de relatie tussen deze biomechanische afwijkingen in het onderste lidmaat en het optreden van PFP nog steeds onduidelijk. De identificatie van deze risicofactoren is echter noodzakelijk om het opstellen van een wetenschappelijk onderbouwde preventie en remediëring van deze pathologie mogelijk te maken.

In de eerste drie studies van dit proefschrift was het de bedoeling om een beter inzicht te krijgen in mechanische intrinsieke risicofactoren ter hoogte van de voet en heup die kunnen leiden tot het ontstaan van het patellofemorale dysfunctiesyndroom. In de eerste twee onderzoeken van dit werk werd op een prospectieve manier nagegaan of parameters van het gangpatroon tijdens wandelen en lopen voorbeschikkend kunnen zijn voor het ontwikkelen van PFP. In het eerste onderzoek werden vierentachtig militaire recruten gevolgd tijdens een zes weken durende militaire basistraining. Voor de aanvang van deze trainingsperiode werd het gangpatroon tijdens wandelen van deze recruten geëvalueerd door middel van plantaire drukmetingen. Tijdens de zes weken durende basisopleiding ontwikkelden zesendertig van de vierentachtig recruten patellofemorale klachten. Uit het onderzoek bleek dat de personen die tijdens initieel voetcontact een meer lateraal georiënteerde plantaire drukverdeling vertoonden en waarbij de verplaatsing van het drukcentrum van de laterale zijde naar de mediale zijde van de voet trager gebeurde tijdens het afrollen van de voet, een hoger risico hebben om PFP te ontwikkelen.

In het tweede onderzoek werd bij beginnende recreatieve lopers prospectief nagegaan of parameters van het gangpatroon tijdens lopen predisponerend zijn voor het oplopen van PFP. Tevens werd bij deze personen de relatie tussen een afwijkende statische belaste voetstatiek en het optreden van PFP onderzocht. Alvorens de start van een tien weken durend ‘start to run’ programma werd bij 102 deelnemende lopers het afrolpatroon van de voet tijdens lopen en de stand van de enkel en voet in stand geëvalueerd. Tijdens het verloop van het ‘start to run’ programma ontwikkelden 17 personen PFP. De resultaten van dit onderzoek toonden aan dat een overmatige verticale piekkracht ter hoogte van de laterale hiel en ter hoogte van de tweede en derde metatarsaal tijdens het lopen discriminerende factoren waren tussen de lopers die PFP ontwikkelden en diegenen die van klachten gevrijwaard bleven. In deze onderzochte populatie werd geen significant bewijs gevonden dat een overmatige gepronkeerde of gesupineerde stand van de voet in stand geassocieerd is met het ontwikkelen van PFP.

In een volgend onderzoek werd getracht een beter inzicht te krijgen in de relatie tussen de kracht van de heupmusculatuur en de beweging van de knie in het frontale vlak tijdens een voorwaartse uitvalspas. Hiertoe werd bij 84 gezonde, asymptomatische personen de isometrische kracht van de heup flexoren, extensoren, abductoren, adductoren, endorotatoren en exorotatoren gemeten. Vervolgens werd bij deze personen de maximale valgus- of varushoek in de knie gemeten tijdens de uitvoering van een voorwaartse uitvalspas. De statistische analyse toonde geen significant verband tussen de kracht van de geteste heupspieren en de hoeveelheid knievalgus of –varus tijdens de opgelegde beweging.



In de vierde studie van dit proefschrift werd nagegaan of variaties in proprioceptieve input ter hoogte van de knie door middel van de applicatie van een strakke kniebrace en matig strakke kous rond de knie tijdens kniebeweging een invloed hebben op de mate van activiteit in de hogere sensorimotorische controlecentra in de cerebrale cortex. Een verbetering van de proprioceptie wordt gesteld als een mogelijk mechanisme waardoor bracing een invloed zou uitoefenen op het verbeteren van de klachten bij PFP-patiënten. Tot op heden is het echter nog onduidelijk op welk niveau van het centraal zenuwstelsel de processing van deze additionele proprioceptieve signalen ter hoogte van de knie wordt geregeld. Hiertoe werd er een MRI studie opgezet waarbij bij dertien gezonde, rechts dominante, vrouwelijke vrijwilligers de mate van hersenactiviteit tijdens de uitvoering van een flexie-extensiebeweging van de rechter knie werd vergeleken tussen drie verschillende condities: 1) met de applicatie van een strakke brace rond de knie, 2) met de applicatie van een matig strakke kous rond de knie en 3) zonder applicatie van een brace of kous rond de knie. De resultaten van deze studie toonden significant meer activiteit in de primaire sensorimotorische cortex tijdens de kniebeweging met applicatie van zowel een brace als kous in vergelijking met de situatie waarbij geen brace of kous rond de knie werd aangebracht. Er werd eveneens een hogere corticale activatie in dit gebied gezien tijdens het dragen van de kniebrace in vergelijking met de conditie met de kous rond de knie. Uit de resultaten van dit onderzoek kan worden geconcludeerd dat de primaire sensorimotorische cortex betrokken is in de centrale verwerking van proprioceptieve informatie, afkomstig van de knie.

Dit doctoraatsproject draagt bij tot een verruiming van de kennis betreffende de rol van alignementbeïnvloedende intrinsieke risicofactoren in de etiologie van PFP. De resultaten van een aantal onderzoeken uit dit proefschrift contrasteren echter met sommige gangbare opinies in de literatuur met betrekking tot de invloed van bepaalde alignementgerelateerde afwijkingen op het ontstaan van PFP. Verder prospectief onderzoek is daarom noodzakelijk om een verhoogd inzicht te krijgen in de rol van deze risicofactoren in de ontwikkeling van het patellofemoraal dysfunctiesyndroom.



